Arizona Medicine

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ARIZONA MEDICINE

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Organization PAGE

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Norman A. Ross, M.D.

doc-tor (dok'ter), n. (Lat. — docere (doctus), to teach).

"***in many respects the conduct of school affairs represents democratic **self-government** at its best.***"[1]

FEDERAL-GRANT-IN AID: SCHOOLS

THE nationwide educational study of the President of the United States, the White House Conference on Education, developed in Arizona under the direction of our Governor. This "grass roots" social review is in preparation for a broad tax recommendation that is to be presented to the Congress of the United States in its coming session. Our profession is second only to the teaching profession in its effect on education. What is your authority and what do you know about past, present, and future grant-in-aid programs? Do grant-in-aid programs apply to medicine directly?

What was the experience of your school with Public Law No. 815?[7]. What is the proposal of the present administration in Washington as interpreted by Smith and Frelinghuysen, S.968, HR 3770?[4] What is the bi-partisan Kearns-Bailey-Humphrey-Ives Bill and proposal, HR 14-15, S. 480?[5] Does your own school need and do you favor federal grant-in-aid (matching funds) for additional classrooms — for your own school's operation and maintenance?

It would appear that both parties in government are convinced that there is to be a great increase in school attendance for the next ten years, that added classrooms are now needed, and that the least painful way of obtaining the necessary additional monies from taxation should be selected. The proposal of the Administration is interpreted as that of encouraging the use of school district and state money by relaxing at local levels of indebtedness controls. The proposal of the bi-partisan group is that of the Federal Government matching local tax and bond money in a manner similar to public health and highway programs.

Those in favor of the administrative proposal fear the loss of community and state independence by continued federal participation. Those advocating the bi-partisan proposal advise that Public Law No. 815 has been used, though it was an outright grant, as a supplement to local funds to stimulate school construction, and the operation of this law has established a pattern in which there is no danger of such a consequence. They state that because of the greater taxing powers, the inequitable distribution of wealth between communities and states, and the fact that the local school district is taxed to its ultimate, that the program of the administration can mean nothing other than delay in solving a problem that is most vital to the national welfare and security.

"***School financing today, because of many factors inherent in the times, presents great difficulties. But the problem should not obscure these basic premises about American education: That adequate education of all American youth is essential to the nation's preservation; that the country's most important resource lies in its citizens — more than in its soil or climate; that every American child has a right to an adeuate educational opportunity; that the American people need an ever-improving educational system, and that they can afford its cost.***"[2]

"***It is safe to assume that the American people do not desire to deny a defensible minimum education to millions of children. Neither do they want to starve thousands of units of our great system of public education into mediocrity. The local-state-federal sharing of costs necessary to prevent such neglect of the national welfare and security must be achieved.***"[3]

"***One little understood aspect of the current situation is the way even those state and local funds which the schools might otherwise have are skimmed off by matching funds offered in huge amounts by the federal government for public assistance, health and rehabilitation services. These federal matching funds tap the national income through the federal income tax and **drain off state and local funds to match them.** The schools lose both ways; first, because they receive no federal funds; secondly, because state and local funds the schools would otherwise receive must be used to match federal funds for other services.

Let us illustrate how this federal discrimina-

tion against education works. Let us think of a local government unit of low economic capacity in which public services of all kinds are needed. Suppose this governmental unit were able to float $500,000 worth of bonds for capital improvements of one kind or another and that hospitals, roads and schools are competing for the funds. Under the present federal laws, the choices are a $1,500,000 hospital, $1,000,000 worth of roads, or a $500,000 school. This is because the **federal government will match the hospital funds 2-1, the highway funds on a 50-50 basis, and the school funds not at all.** Needing all of these facilities, the federal incentives against the use of limited state or local funds for construction of schools are strong indeed.

But even this is not the worst feature of the federal discrimination against education in favor of highways and other services. In case of economic depression, federal funds would preserve matched funds and the **schools would be the first to be cut.** They must be placed in a position of equality with other public services so that they can compete equally for state and local funds without both direct and indirect discrimination against them by federal matching funds favoring their competitors.***[3]

"***The type (of legislation) approved by experts in educational finance, education associations and most citizens' groups is represented by the bi-partisan Kearns-Bailey-Humphrey-Ives proposal (H.R. 14-15; S. 480). These and similar bills would authorize federal funds for the states according to the number of children 5-17 years of age. The state educational agencies would allocate the funds to local school districts according to their relative needs, fiscal abilities and past and present efforts to construct schools for themselves. The priorities among school districts defined by each state would make certain that the federal funds are allocated for school construction that would not otherwise occur. School districts fully competent to construct their own schools would not be eligible for federal funds.

Under this type of legislation the states would account to the federal government for the federal funds. School building standards would be left to the states and localities. There could be no federal control of educational programs conducted in the buildings after they have been completed. This bi-partisan legislation has been considered repeatedly in Congressional hearings for four or five years. The bill sponsored by Senator Lister Hill[6] and several other senators differs only in minor details.***[3]

"***I do not favor a permanent federal school building program. Once we have stimulated construction and eliminated the present classroom deficit, it is my feeling that such a program, should be terminated.***"[1a]

"***If we are forced to place priorities and choose this year between a national highway program and a national classroom construction program, I shall cast my lot with the schools."[1]

The Editor-in-Chief has suggested that this page could treat with controversial matters. This paper includes numerous quotes and quotes from content. Contrary to what you may now think, two of those authorities quoted arrived at conclusions which were in some instances directly opposed. To know your school community, to understand the proposed Federal grant-in-aid programs, we refer you to the complete articles. Another excellent source of information is the National Citizens Commission for the Public Schools, 2 West 45th Street, New York 36, N.Y. Should you express your interest to this group you will receive regularly "Better Schools", their publication.

References and sources of material for this paper are as follows:

1. Representative Stewart L. Udall, Democrat, 2nd Congressional District, State of Arizona, Congressional Record Appendix A 1712.

1a. Representative Stewart L. Udall, personal letter.

2. Roger A. Freeman, Assistant to Governor Langlie of Washington, Research Head for the Commission on Inter-Governmental Relations Committee on Federal Responsibility in the Field of Education, "The Challenge of Federal School Bill Aid" as published in the Newsletter of the Arizona Tax Research Association, July, 1955.

3. Dr. Edgar Fuller, Executive Secretary, Council of Chief State School Officers, formerly President of Eastern Arizona Junior College, a manuscript entitled WHY THE FEDERAL GOVERNMENT MUST SHARE IN THE FINANCING OF PUBLIC SCHOOLS, published in the Rotarian, 4-11-55.

4. S.968, HR 3770, Smith and Frelinghuysen. Senator H. Alexander Smith, Republican, New Jersey and Representative Peter Frelinghuysen, Jr., Republican, Fifth District, New Jersey, are authors of this legislation which is considered to be their interpretation of the Administration's approach to a grant-in-aid program for school construction.

5. HR 14-15, S.480, Kearns-Bailey-Humphrey-Ives. Representative Carroll D. Kearns, Republican, Twenty-Fourth District, Pennsylvania. Representative Cleveland M. Bailey, Democrat, Third District, West Virginia. Senator Hubert H. Humphrey, Democrat, Minnesota. Senator Irving M. Ives, Republican, New York.

6. Senator Lister Hill, Democrat, Alabama. He is co-author of the Hill-Burton Federal Grant-in-Aid Bill for Hospitals, and has been an important factor in the Senate proposing school legislation.

7. Public Law No. 815. This law provided for Federal grant-in-aid for school districts for classroom construction. Federal money was an outright grant. No matched state or local funds were required. This law gave relief to school districts that had increased enrollment resulting from war migration.

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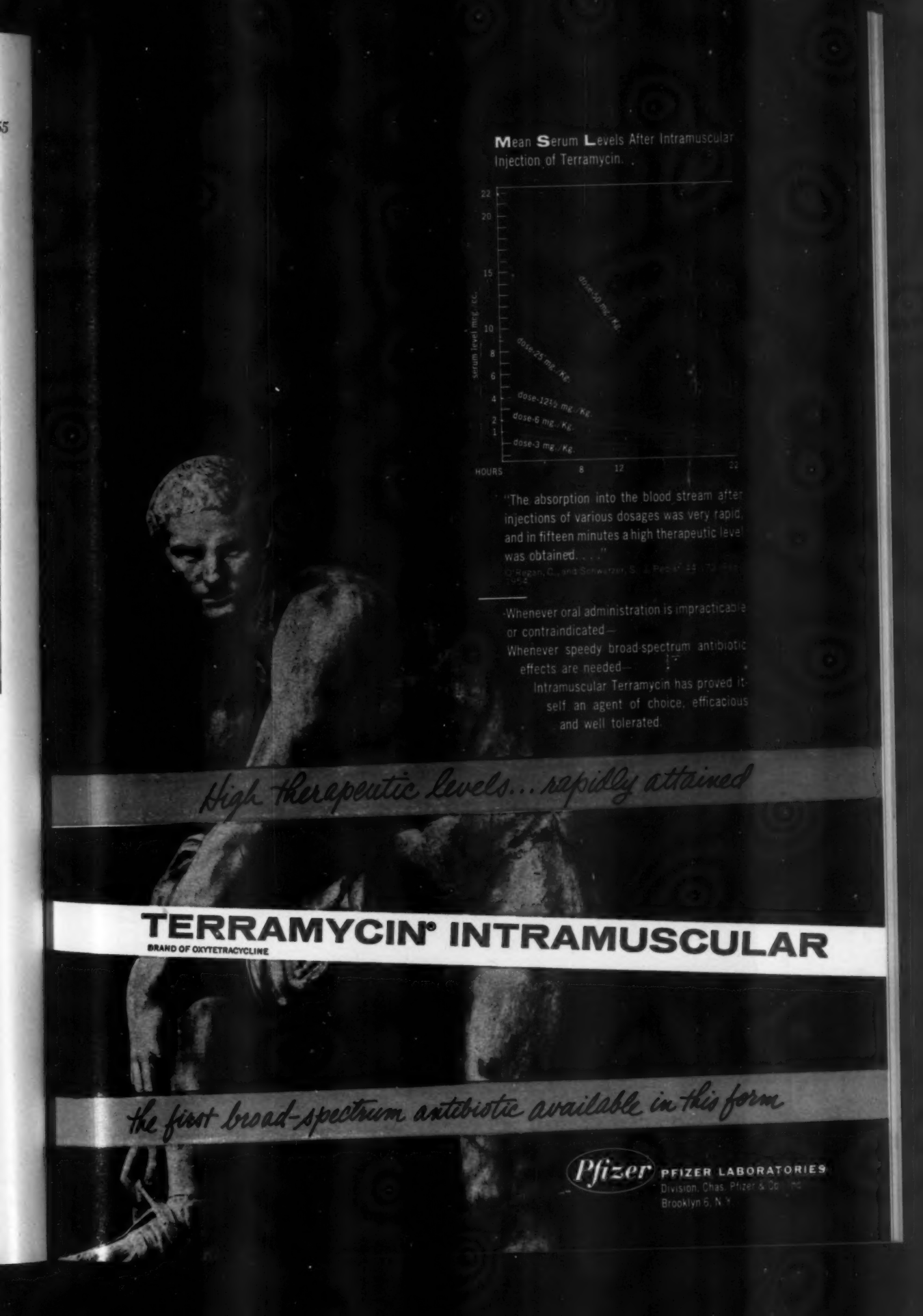
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20
15
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2
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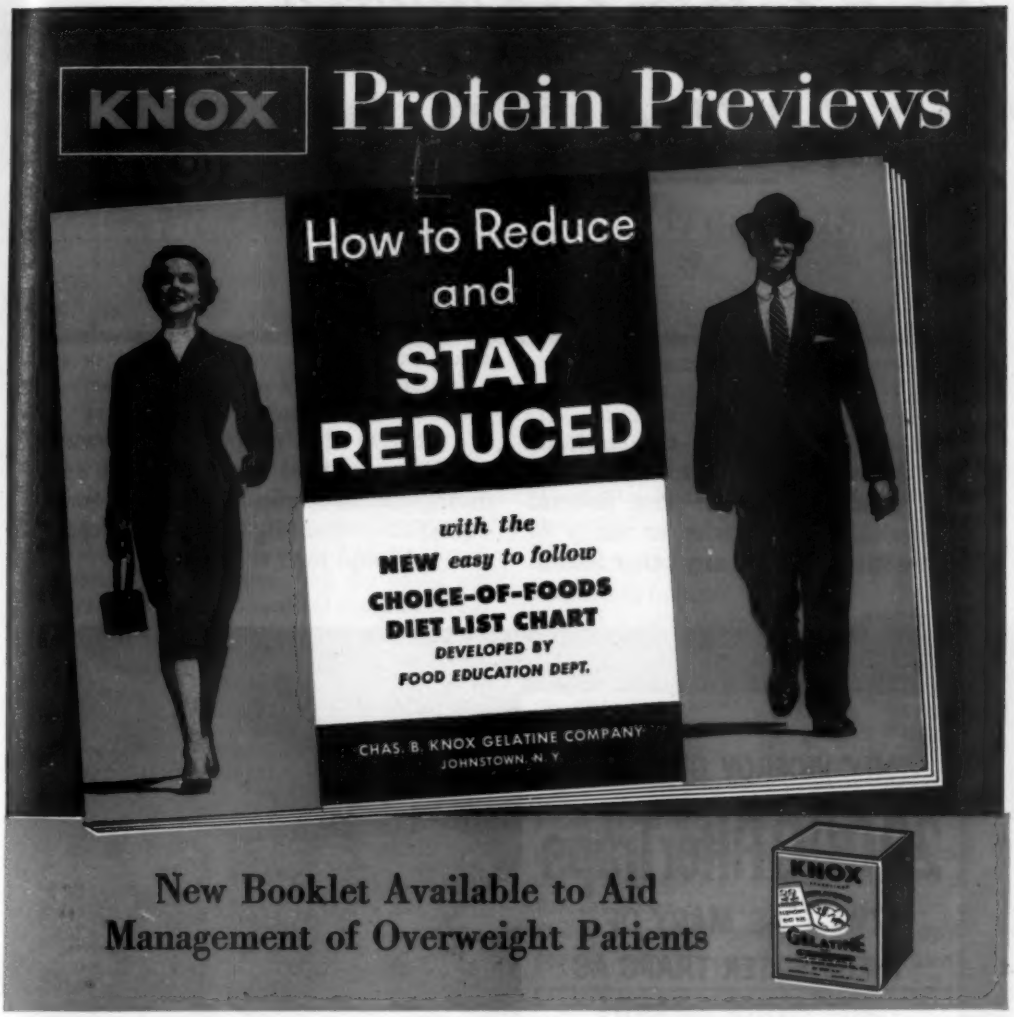

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ITEM	WEIGHT OF SERVING gm.	PROTEIN gm.	ENERGY calories
Fruit	77	0.4	68
Cereal (dry wt.)	30	3.2	110
Enriched White Bread (toasted)	50	4.2	130
Sugar	10	0.7	40
Butter	10	0.1	73
Whole Milk	480	16.9	330
Calories 750		Fat (gm.).....	28
Protein (gm.)..... 25		Carbohydrate	100

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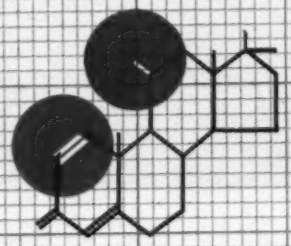

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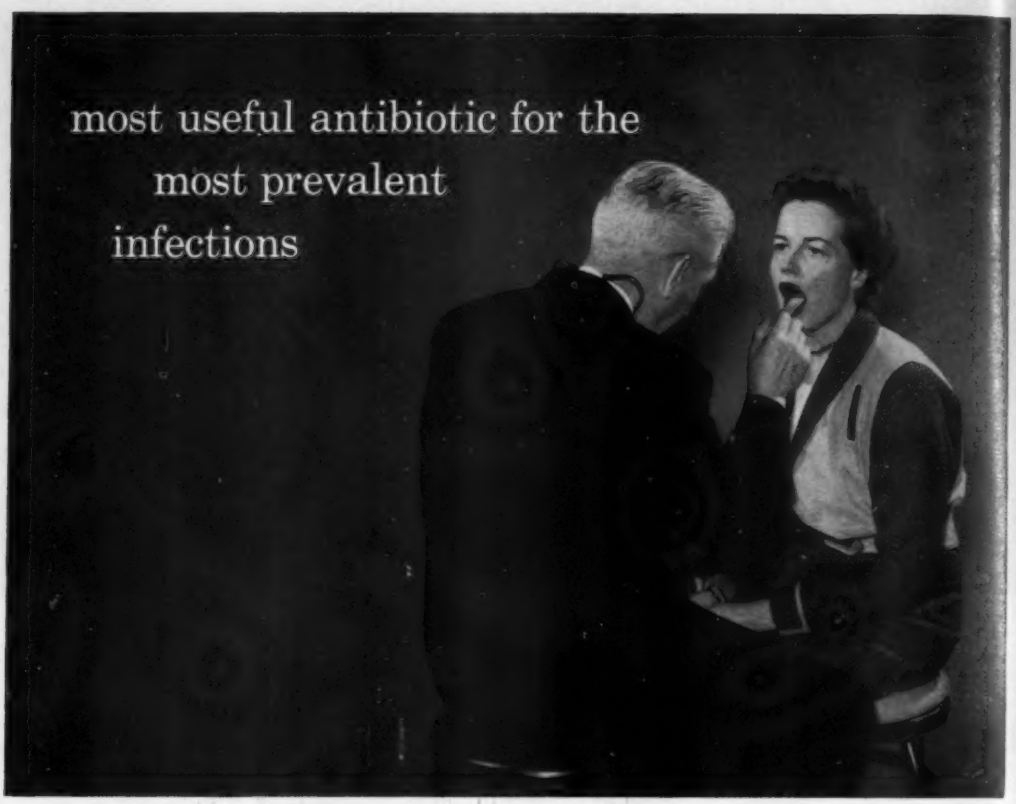

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Original ARTICLES

THE PREVENTION OF RECURRENT URINARY CALCULI

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Phoenix, Arizona

AFTER a long era of unintentional neglect, the medical profession has become aware of the fact that the formation of urinary stones is a symptom and not a disease. And with this awareness has come the realization that our obligation to the patient does not terminate when the offending calculus has been removed. Rather, our responsibility is just beginning, for we currently have at hand measures which, though not a guarantee against stone formation, will do much to minimize the possibility of recurrent stones. The value of obviating the attendant misery to the patient and the danger to functioning renal tissue is perspicuous. It was with the hope of reviewing some of the acceptable and effective anti-stone regimens that this paper was prepared.

Though urinary calculus disease is an ubiquitous entity, it is well known that Arizona shares with Southern California and Florida the dubious distinction of belonging to America's Stone Belt.(1). Because of this, it is probable that in Arizona more patients with urinary tract stones are seen than in some other sections of our country. It is also accepted that recurrent stones are more frequently observed in this area as the factors tending to produce the initial stone are usually still prevalent after its removal. The significance of this should be emphasized, for the salient long range factor in the battle against stone recurrence concerns the preservation of renal function. With the spontaneous or surgical removal of every stone, alterations of major or minor degree take place in the kidney which, in addition to reducing renal function, also predisposes to the subsequent development of further stones. We are all familiar with the end stage in extensive stone disease with the tragic appearance of ever increasing azotemia, or with stone damage so extensive that good health and sometimes life are not sustainable.

The aforementioned neglect of the past does not merit lengthy consideration. It is well to recall that in analyses by Higgins and others, recurrent stones occurred in 16.9 per cent, and if only those cases were considered in which urinary infection was present, this figure of recurrence was as high as 30 per cent. It is true that measures were not at hand during that period of observation as effective as those available to us now. But more responsible for this unacceptable recurrence rate was the fact that the philosophy in the past had been to remove the stones that were present and then passively await the development of others. Unfortunately, all too often, there was no disappointment in this sense.

A scientific approach to stone formation was preceded by rational but often ineffectual measures. These included the acid ash or alkaline ash diet and increased fluid intake. It was not until accurate methods of quantitative analysis of the components that form stones as seen in the serum, stool, and in urine were available that well founded preventative diets were developed. Equally pertinent was the development of accurate methods for the analysis of the nidus

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of the stone. By this means it is possible to identify the initial crystalloid substances which coalesce to form a stone, and to propose methods to prevent the initial precipitation of this crystalloid.(2)

The actual physico-chemical processes involved in the formation of a renal stone comprise a polemic subject. Whether the initiating "seed" develops as a subepithelial crystal which causes erosion of the epithelium over a renal papilla or whether it is simple precipitation of crystalloids from a supersaturated solution is not conclusively established. Nonetheless it is known that if a situation exists, either in the collecting tubules or calyceal system, in which the crystalloid-colloidal baalnce is subjected to greater saturation than can be compensated, precipitation and subsequent stone formation will occur. Such a situation is present in the development of calcium phosphate stones when calcium excretion is greatly increased for one reason or another. Marked increases in calcium excretion in hyperparathyroidism, idiopathic hypercalcinuria, vitamin D intoxication, recubency, postmenopausal osteoporosis, cortisone therapy and so forth, are familiar to all of you. Such conditions also result in calcium carbonate stones. When infection is present, especially with urea splitting organism, magnesium ammonium phosphate stones form rapidly. The altered metabolism resulting in increased urate or cystine excretion acts in a similar manner to produce stones of corresponding composition. (See Table 1)

It is immediately apparent that several simple measures will be of aid in the patient who has had a urinary stone. By increasing fluid intake, the resultant urine will be correspondingly more dilute. Hence urinary crystalloids such as calcium phosphate, uric acid and so on, will be less saturated and less likely to precipitate. Vermuelen, Goetz and co-workers, using zinc discs in rat bladders demonstrated quantitatively the much more rapid growth of stones on these discs with conditions of dehydration.(3). The benefit of this simple feature is unquestioned but great excesses of water often are intolerable to the patient because of anorexia and other side effects. Depending on the type of stone, an additional method of management includes the use of an acid ash or alkaline ash diet. By thus altering the urinary pH, the solubility of any stone forming substance may be enhanced. Needless to say, many other factors such as adequate vitamin A to prevent keratinization of the uroepithelium, correction of strictures and vesical neck obstruction to obviate stagnation of urine, and attempts to control infection have all served a useful purpose in the past.

Three recent methods to aid in stone prevention will be briefly reviewed with their attendant rationale. All are utilized as adjuncts to the more conventional high fluid and dietary programs just discussed. The first will be mentioned only for the sake of completeness. In the prevention of calcium stones it was found that citric acid formed a very soluble substance when it combined with calcium. Unfortunately, ingested citric acid is almost completely metabolized. However, estrogenic substances were found to increase markedly the endogenous production and excretion of citric acid. This method has not been widely accepted as the dosages of female hormones required are sufficient to produce menstrual changes in women and feminization in men.(4) Of the two most widely heralded anti-stone programs, the first concerns the usage

TABLE 1

ETIOLOGICAL FACTORS IN FORMATION OF STONES OF VARYING CHEMICAL COMPOSITION

Chemical Analysis of Stone Nidus	%	Reaction of Urine	Accepted Etiological Factors
Calcium Phosphate	61	Slightly acid to slightly alkaline	Hypercalcinuria: High calcium intake; Recumbency; Vitamin D intoxication; Infection; Post-menopausal osteoporosis; Ideopathic hypercalcinuria; Hyperparathyroidism.
Calcium Carbonate	4	Neutral to slightly alkaline	Same as for calcium phosphate
Magnesium ammonium phosphate	Usually secondary	Alkaline	Urinary infections especially with urea-splitters; Presence of calcium phosphate nidus; High phosphate excretion.
Calcium oxalate	23	Neutral to slightly alkaline	High calcium and oxalate intake; Dehydration; Nidus of calcium phosphate
Uric acid	10	Acid	Gout; Dehydration.
Cystine and xanthine	1	Acid	Familial defect (Metabolic defect)

of hyaluronidase. It has long been postulated that crystalloids in the urine which are normally in a supersaturated solution, are prevented from precipitating by their adherence to urinary colloids. These colloids, carrying similar electric charges, act to repel other colloidal particles and thereby prevent conglomerates from forming. Dr. A. J. Butt and his co-workers have studied the dispersing powers of such colloids and found them to be especially abundant in pregnant women and Negroes – people in whom the incidence of stone formation is low. On the contrary he found that stone formers had reduced urinary colloids. He then proposed to increase the colloids in these people by injecting hyaluronidase subcutaneously at regular intervals. This substance acting upon the hyaluronic acid component of intracellular ground substance produces products which are excreted in the urine and have a strong peptinizing and dispersing action. The addition of these colloids acts to enhance protective colloidal action. By so doing he has shown with the electron-micriscope ultracentrifuge and also clinically, that stone formation is substantially depressed. (5). A gross examination of the patient's urine shows clearing of the turbidity usually seen in stone formers and reduction of crystalloid sediment.

Ephraim Shorr is credited with the second widely used anti-stone regimen. Rationale for his treatment rests in the fact that calcium phosphate is very often the initial crystal forming urinary stones and is believed by many to be the nidus for calcium carbonate, calcium oxalate, and magnesium ammonium phosphate stones. In an effort to reduce urinary phosphate excretion, he diverted the route of the phosphorus excretion from the urine by giving patients aluminum hydroxide orally. The aluminum combines with the phosphorus in the small bowel to form an insoluble aluminum phosphate which is almost entirely excreted in the stool. This results in a reduced phosphorus assimilation and a resultant hypophosphotemia. Very little prosphorus is then available for the glomerular filtrate and a compensatory increased reabsorption in the distal convoluted tubule takes place. Because of this, phosphorus is not present in significant quantities in the urine for precipitation with calcium, and stone formation is depressed.(6) Table 2 demonstrates this altered route of phosphorus excretion when such a program is instituted.

A review of the literature indicates that good results have been obtained with either method, supplementing the general features previously mentioned. It should be stated that Dr. Butt's results have not at all times been duplicated by others who have used hyaluronidase. Nonetheless, in Dr. Butt's initial series he treated twenty-four patients who had been chronic and rapid stone formers with 150 to 900 turbidity reducing units (TRU) of hyaluronidase every twenty-four to forty-eight hours. In 79 per cent of these (19) there were no new stones that developed nor was there progression in size of existing stones.(7) In four of these nineteen, stones already present appeared to become less dense. It is well to remember that the patients in this study had severe calculous disease and had demonstrated their propensity for stone formation by producing multiple calculi in the past.

Barrett presented a series of thirty-four patients who had had intractable stone disease.(8) He treated these with the regimen proposed by Ephraim Shorr. Using a neutral diet with 0.6 grams calcium, 1.2 grams phosphorus, and 0.2 grams magnesium per day, supplemented by 30 cc. of aluminum hydroxide gel Q.I.D., he was able to prevent formation of any new stones or increment in size of those already present in thirty of the thirty-four patients (88%). The

TABLE II

INGESTION AND EXCRETION OF SOME COMMON STONE CONSTITUENTS

Element	Mgms. Ingested Daily	Normal Serum Value (mg. %)	Mgms. Excreted Daily in Stool	Mgms. Excreted Daily in Urine
Calcium	700-900	9-11	500-600 (70%)	100-200 (30%)
Phosphorus	1200-1400	3-5	300-500 (30%)	1000 (70%)
Magnesium	500	1.9-2.1	100-200 (35%)	300-400 (65%)
EFFECT OF BASALJEL REGIMEN ON PHOSPHOROUS METABOLISM				
Patient receiving 120cc. of Basaljel daily with identical diet				
Phosphorous	1200-1400	3-5	1000-1300 (85%)	100-350 (15%)

period of observation extended over a period of twenty-one months. Undesirable side effects were few, manifesting themselves as mild anorexia or constipation.

In the past years, in an effort to evaluate the recently presented anti-stone programs, we have had the opportunity of following sixty-one patients who have had three or more stones develop in the five year period prior to our period of observation. All had been on conventional treatment consisting mainly of high fluid intake and some dietary restrictions. Because stones continued to be formed, they were considered suitable for our study. A larger group had been thoroughly evaluated and the presence of any obstruction, metabolic disorder, or infection determined. If any such feature was detected, the patients were excluded from the study. There remained sixty-one patients who had no primary cause for stone formation. Thereby, they represented that largest group – the idiopathic stone formers. They were then placed on varying programs. All were given fluid from 3500 to 4000 cc. of distilled water daily. All received multiple vitamins, all were well mobilized and maintained on a standard diet.

The first group of thirty-six patients received 30 cc. of aluminum hydroxide gel four times a day. Metabolic studies were done to determine the extent of reduction of phosphorus assimilation due to its precipitation with aluminum in the bowel. It was seen that urinary excretion of phosphorus could be reduced from a normal of about 1000 mg. daily to 150 to 350 mg. a day. Such a reduction did not interfere with the bionic state for the excellent factors of physiological economy, maintained positive phosphorus, calcium, and nitrogen balance. These 36 patients had had stone problems for an average of 7.9 years. In the past they had formed an average of 4.8 stones. Aluminum hydroxide gel was continued for an axerage at 21 months. We might have expected them to form 43 stones had these patients continued as their past records would indicate in this 21 month period of observation. Instead, only 11 stones appeared while under therapy, a conclusive decline in calculus formation.

Thirteen other patients had experienced the problem of forming urinary stones for an average of 6.1 years. In this period they had produced an average of 4.1 stones. This group was treated with hyaluronidase 150 to 750 T.R.U. every forty-eight hours for an average of fourteen months. Examinations of urine specimens to assure clearing of all turbidity were performed daily on some patients to evaluate grossly the effectiveness of this enzyme. In this group, seventeen stones were statistically anticipated and only eight (or less than 50%) appeared.

A final group of twelve patients who had less severe calculous disease for an average of 4.9 years (and had had an average formation of 3.9 stones in this period) were treated by intensive urinary acidification. Using sodium acid phosphate gr.10 Q.I.D. and careful appraisal with a Lamotte indicator, the pH was maintained between 5.5 and 6.0 as much as possible. Again the anicipated stone formation was cut in half with only eight stones foring against a calculated sixteen. It would be timely to indicate that though acidification programs are the most easily tolerated, they are not always feasible. Their rationale is based on the fact that the solubility of stones such as calcium phosphate doubles with each 0.5 unit reduction in pH (within the usual urinary range of 5.0-7.0). The efficacy of this hydrogen ion alteration is therefore apparent. However, such programs are contraindicated if impaired renal function has already been accompanied by acidosis. Secondly, in severe stone problems in which refractory urinary infections as with Proteus and other urea splitters is present, the urine is often in the acid range only in the collecting tubules. Upon entrance into the infected renal pelvis, the excessive ammonia formation by the bacteria makes acidification an impossibility. In such instances the increased mobilization of calcium produced by acidification may, in conjunction with the alkalinity of the infected renal pelvis, actually act in deleterious manner and enhance the formation of stones. Schematic data demonstrating the success and failure to produce acidification in sterile and infected urines respectively is shown in Figure 3.

From the results in the literature and in our own study, it is apparent that attention to diet, supplemented by one of the new and rational stone programs will substantially reduce the severity of this disease. Other proposed regimens utilizing enzymes such as urease to break up the stone matrix have not been evaluated. Table 4 summarizes the results of our study.

Most significant is the realization that much can be done to aid the recurrent stone former.

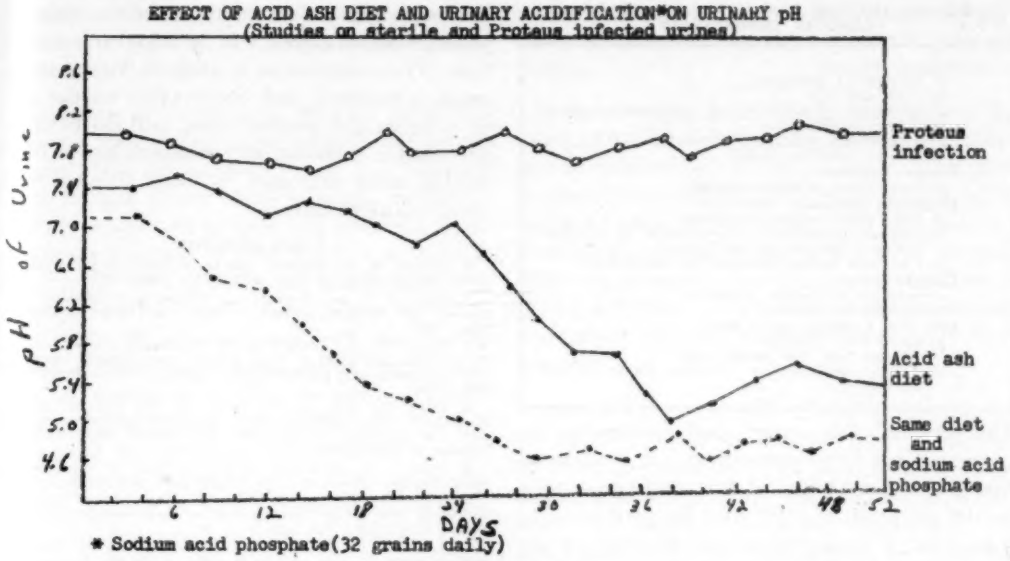

TABLE IV

RESULTS OF VARIOUS ANTI-STONE REGIMENS IN 61 CHRONIC STONE FORMERS

Type of Regimen	No. of Patients	Ave. Duration Calculous Disease	Aver. No. of Previous Stones	Ave. Duration of Regimen	Ave. No. Stones Formed During Regimen	Ave. No. Stones Formed During Similar Period Before Therapy
Aluminum hydroxide	36	7.9 yrs.	4.8	21 months	.3 per patient (11 stones)	1.2 per patient (43 stones)
Hyaluronidase	13	6.1 yrs.	4.1	14 months	.6 per patient (8 stones)	1.3 per patient (17 stones)
Uurinary acidification	12	4.9 yrs.	3.9	20 months	.7 per patient (8 stones)	1.4 per patient (16 stones)

Best results are obtained on rigid management that renders every opportunity to avoid further calculous disease. General measures include high fluid intake, adequate vitamin ingestion, absolute control of infection and relief of obstruction, active mobility, and frequent follow ups. Specific therapy depends upon the type of stone disclosed by chemical or diffraction analysis. Calcium phosphate, calcium carbonate, magnesium ammonium phosphate and calcium oxalate respond best to urinary avidification or aluminum hydroxide gel or possibly both. It is implicit that the more seriously the condition threatens the physical integrity of the patient, the more vigorous and stringent will be the therapeutic efforts. Uric acid and xanthine stones are best prevented by a restricted protein, alkaline ash diet supplemented by a substance such as sodium citrate four grams Q.I.D. to alkalinize the urine. In particularly difficult problems, hyaluronidase might also be employed in these cases. With careful observation and patient surveillance, good results may be anticipated for any rational regime. Suggestions for prevention of recurrent urinary stones are outlined in tables 5 and 6.

TABLE V
GENERAL PRINCIPLES IN ALL ANTI-STONE REGIMENS

1. Fluids to 3500-4000 cc. daily. (Distilled or boiled)
2. Multiple vitamin tablet twice daily.
3. Correction of all obstructive features.
   a) No residual in bladder.
   b) No retention in renal pelves.
4. Absolute control of infection.
   a) Culture and sensitivity studies.
   b) Intermittent antimicrobials.
5. Early mobility.
6. Persistent and prolonged surveillance.
   a) Microscopic urinalysis every 2 months.
   b) KUB every 6 months.
   c) Daily pH determinations by patients.
7. Chmical analysis of stone nidus.
8. Dietary restriction as indicated by stone analysis.

Summary: The significance of recurrent stone formation and some of the more recent concepts concerning prevention have been reviewed.

TABLE VI

SPECIFIC THERAPY FOR STONES OF VARYING CHEMICAL COMPOSITION

1. **Calcium phosphate**
   a) All general principles.
   b) Acid dish diet.
   c) Restriction of calcium and phosphorous intake.
   d) Urinary acidification.
      (Soduim acid phosphate gr. x Q.I.D.)
   e) Basaljel (15-30cc. Q.I.D.)
2. **Calcium carbonate**
   a) Same as for calcium phosphate.
3. **Magnesium ammonium phosphate**
   a) Same as for calcium phosphate.
   b) Particular attendtion to eradication of infection.
      (Use of Furadantin for Proteus)
   c) Early and intense treatment for urea-splitters.
4. **Calcium oxalate**
   a) Same as for calcium phosphate.
   b) Diet low in oxalates.
5. **Uric acid, Xanthine, and Cystine**
   a) All general principles.
   b) Alkaline ash, low protein diet.
   c) Urinary alkalinization. (Sodium citrate 3 gms. Q.I.D.)

The proposal that many renal stones can be prevented has been entertained. It is assumed that the responsibility of this prevention rests on the physician to meet with his patient. The principles of management are familiar as are the facilities for diagnosis and stone analysis to determine which method is most suitable for each patient. Few patients who have suffered the excruciating discomfort attendant upon repeated urinary stones will be found uncooperative. The combination of analysis, logical therapeutic principles, and cooperation on the part of patients and medical men, will do much to prevent the suffering and resultant loss of functioning renal substance to those afflicted with renal calculous disease.

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PERNICIOUS ANEMIA WITH THE ABSENCE OF TYPICAL ANEMIA

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IT is the purpose of this paper to call attention to the fact that currently one may encounter many cases of neuropathy with achlorhydria without the typical hematological picture of pernicious anemia, whose proper diagnosis and treatment should not be missed because of the absence of characteristic peripheral blood and bone marrow findings. This fact has been mainly brought about since the advent of popular mass consumption of vitamins,(1) especially folic acid and B_{12}, and apparently also antibiotics. There is no doubt that oral intake of folic acid, 5 milligrams daily, effects a hematologic remission while the spinal cord lesions are aggravated in pernicious anemia. It was definitely shown that folic acid requires no intrinsic factor and has a direct action upon the bone marrow, converting a megaloblastic into a normoblastic bone marrow. Vitamin B_{12} plus intrinsic factor, incorporated into a host vitamin preparations on the market, when taken orally tend to suppress megaloblastic erythropoiesis while being insufficeint to cause amelioration of the progression of postero-lateral cord changes for which massive parenteral medication is required. The mechanism of action of penicillin or tetracyn is as yet not with certainty confirmed, but apparently they effect increased intestinal absorption of Vitamin B_{12} by increasing its biosynthesis. Hence in the present era of mass production and widespread ingestion of those medications combined with the relatively high sensitivity and prompt response to them of the hematological apparatus in contradistinction to the resistant neurological system, the number of patients visiting for the first time a physician's office and presenting abnormal neurological manifestations without an abnormal blood picture will become more frequent. Thus a peculiar phenomenon will come to the fore: A disease called pernicious anemia representing itself without anemia. But this is the challenge the physician will have to be faced with today: That not the name, per se, of a disease but rather the disease itself, in all its manifestations, should serve to command proper mobilization of diagnostic skills, has been a not uncommon experience in clinical medicine.

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Suffice it to mention aortic insufficiency and calcification of the ascending aorta with no positive serology for syphilis, or diabetes mellitus without mellituria.

Awareness of dissociation between hematological and neurological improvement was known already(2) in the previous era of liver therapy, when it was noted that subacute combined sclerosis progressed in a patient who had taken inadequate amounts of liver which were enough to bring the red cell count and bone marrow to normal. However, at no time before were serious bodily insults associated with the disease process of pernicious anemia apt to escape diagnosis and treatment as in the present era of antibiotics, folic acid, oral vitamin B_{12} plus intrinsic factor, and parenteral B_{12} inadequately administered, mostly empirically, in a host of conditions; all leading to the more frequent occurence of normal hemopoiesis in cases that happen to be afflicted with pernicious anemia. In addition to the above it appears that many a physician has been overtaken by the textbook picture of a patient who reveals pallor or lemon-yellow tint, weakness and fatigue, palpitation and dyspnea, and even signs of renal or cardiac failure in association with anemia; macrocytosis, hyperchromia, poikilocytosis, Howel-Jelly bodies, basophilic stippling, Gabot's rings; leukopenia with neutrophilic multisegmentation; thrombocytopenia, and a megaloblastic bone marrow. *All of these symptoms and signs associated with anemia, however, including leukopenia and thrombocytopenia, appear to be doomed to play a lesser role in elicitation of a diagnosis of pernicious anemia as compared to subacute combined sclerosis in association with histamine refractory achlorhydria.* The latter sign is of utmost importance because of its resistance(3) to even massive parenteral doses of liver or B_{12} and also because of its being associated steadily with the deficiency of intrinsic factor which is the basic etiological agent responsible for the development of pernicious anemia.

The incidence of subacute combined degeneration of the spinal cord with histamine-resistant achlorhydria in association with other diseases except pernicious anemia is extremely rare,(4) and when encountered can be distinguished on clinical evidence alone. Here may be mentioned sprue(5), diabetes mellitus, pellagra(6) and mainly tapeworm(7) infestation as well as gastric carcinoma(8), post gastrectomy syndrome(9), CA of the colon and strictures of the small bowel or multiple intestinal fistulas.

A question may arise, though, especially in the mind of a thorough investigator, whether there are, in addition to clinical evidence, other procedures that could be successfully employed in order to confirm specifically the diagnosis of pernicious anemia in the absence of anemia. Before an answer to this question can be discussed it is felt worth while to review the physiologic and other changes associated with pernicious anemia.

PHYSIOLOGIC AND OTHER CHANGES IN PERNICIOUS ANEMIA: Castle(10) stated that "the disease would not develop if the patient could effect daily the transport of a millionth of a gram of vitamin B_{12} the distance of a small portion of a millimeter across the intestinal mucosa and into the blood stream. This he cannot do, principally as a result of failure of the stomach to secrete into its lumen some intrinsic factor. The cause of the stomach's failure to secrete the intrinsic factor is not known or idiopathic and hence the name "primary" pernicious anemia.

In human serum vitamin B_{12} consists of two forms, the one which is protein bound, and the free form. In normal subjects the mean total vitamin B_{12} was found to be about 400 millimicrogram, and free vitamin B_{12} about 70 millimicrogram per milliliter of serum; while in a pernicious anemia patient in relapse no free vitamin B_{12} is detected and and total vitamin B_{12} concentration is about 40 millimicrogram per milliliter, or 10% as much as in a normal person. The liver(12) in an untreated pernicious anemia patient contains no vitamin B_{12}, while the liver of a normal person is estimated to carry about 200 microgram of vitamin B_{12}. Gridwood reported that assays of tissues for vitamin B_{12} in patients with pernicious anemia in relapse showed no detectable amount of B_{12}, whereas corresponding normal tissues contained measurable quantities.

The absence of free vitamin B_{12} in the blood and of total vitamin B_{12} in the liver and tissues, and the presence of only one-tenth of normal concentration of total vitamin B_{12} in the serum of a pernicious anemia patient in hematological

relapse, is due to intrinsic factor deficiency in the gastrointestinal tract preventing vitamin B_{12} absorption into the blood stream and causing its loss in the feces.

Normally, following absorption, protein bound vitamin B_{12} rapidly appears in the plasma from which it is transported to various body tissues. Most investigators(14) except Horrigan and Heinle, believe that the factor contained in the serum which exerts a binding effect on vitamin B_{12} shows the same binding capacity in normal as well as in pernicious anemia cases.

Lajtha and associates(15) found that neither B_{12} nor intrinsic factor separately exerted any effect on cultures of bone marrow cells from patients with untreated pernicious anemia, but in combination they stimulated the maturation of the megaloblasts. Serum of normal subjects exerted a similar effect, whereas serum of untreated cases of pernicious anemia had an inhibitory action. If B_{12} was administered parenterally to such patients, their serum stimulated maturation of the cells, like normal serum. Lajtha concluded that B_{12} must have combined with "intrinsic factor" present in the body tissues or fluids in order to have acquired such activity.

Thus it is B_{12} that is low or absent in the serum and tissues of the untreated pernicious anemia patient, but not *"tissue intrinsic factor" which is adequately present* in contrast to "gastric intrinsic factor" which is markedly deficient, and is actually responsible for the disease process. In other words, *intrinsic factor has a dual action: "gastric intrinsic factor" effects absorption of vitamin B_{12}, and "tissue intrinsic factor" makes possible B_{12} action upon megaloblasts in the bone marrow causing their maturation or normoblastic conversion.* It appears that the transformation of thymine to a nucleoside, thymidine, which then enters into the formation of nucleic acids needed for maturation of erythrocytes, is a metabolic function of vitamin B_{12} promoted by the "tissue intrinsic factor." That vitamin B_{12} has a definite neurotrophic effect and is essential in the functional integrity of myelinated nerve fibres of the brain and spinal cord is best witnessed clinically by its relationship to subacute combined sclerosis. The role played by "tissue intrinsic factor" in the latter action has never been investigated, nor has anybody tried to determine vitamin B_{12} concentration in the spinal fluid of normal, treated or untreated pernicious anemia cases.

Normal persons have a total of 1 microgram of B_{12} in circulation; pernicious anemia patients in relapse have one-tenth as much. Within one hour after an intramuscular injection of 40 microgram given to a patient with pernicious anemia in relapse the total of B_{12} in the plasma rises to about 3 microgram and falls within 24 hours to approximately 0.75 microgram. In view of the fact that as much as 34 microgram out of 40 microgram injection is not excreted, it appears that the greater part of the dose is stored in the body outside the plasma and that the plasma concentration in patients with pernicious anemia receiving no further treatment is maintained by drawing upon this store(14). A radioactive tracer employed by Glass revealed hepatic uptake to be several times as high as that of other tissues and hepatic decline to be slower than elsewhere in the body. Vitamin levels in the liver rise to peaks four to six days after injection and five to seven days after oral administration. Two or three months later, 85% of peak values are still demonstrable. *Hepatic storage probably accounts for the long period after deprivation before a deficit ensues,* as well as for delayed onset of macrocytic anemia after total gastrectomy. *In pernicious anemia when therapy is stopped it has been reported*(17) *that hematological relapse may not occur for as long as* 3 *years.* The daily human requirement is not more than 1 microgram a day, or less than 0.5 milligram a year. Vitamin B_{12} liver stores in a patient who has had a total gastrectomy may be sufficient for his needs for several years. Poulson and Harvey in studying the effects of total gastrectomy on erythropoiesis in 27 patients, at varying intervals over a decade, found macrocytosis to occur within one or two years after operation, and if the patients lived long enough they developed anemia and still later a megaloblastic bone marrow and, as one of this group did, subacute combined sclerosis. One of their patients also showed signs of jejunal atrophy not unlike gastric atrophy seen in primary pernicous anemia.

In primary pernicious anemia there is a true decrease in the thickness of the mucosa of the fundus(19). The glandular tubules are altered and greatly decreased in number, and tubular epithelium is also strikingly altered. Parietal and peptic cells are rare or absent. There is an increase in interstitial tissue. Atrophy of the sub-

mucosa and muscularis has also been noted. Some areas of the gastric mucous membrane may assume microscopic characteristics typical of the small intestine. No atrophy in either the pyloric region of duodenum is noted. The argentaffine cells are absent. There is increased frequency of both benign and malignant tumors of the stomach. Closely related to the histopathologic changes are the macroscopic findings. Henning, in his monograph, described diffuse atrophy of the entire gastric mucosa with a grayish yellow to grayish green color, especially when the stomach was well inflated on gastroscopic examination. The "sharp outline of distinct fine and coarse blood vessels" could be seen arching over the wall. Schindler(20) did not find invariably complete diffuse atrophy but rather patchy gray areas of atrophy between areas of normal or only very slightly superficially inflamed mucosa. Following treatment with liver or B_{12} the mucosa may be restored in variable degree to a normal gastroscopic appearance, but whether this represents a restitution to histologically normal mucosa has not been demonstrated.

Biochemical changes observed during early remission induced by vitamin B_{12} include the production of positive nitrogen balance (21) of as much as six grams daily and elevation of the serum uric acid and an increase in urinary excretion of uric acid(22). During the peak of reticulocyte response to initiation of B_{12} therapy there is also an increased phosphorus excretion in the urine. These changes may be related to changes in nucleoprotein metabolism which accompany alteration of a megaloblastic to a normoblastic bone marrow. The iron content of the serum as well as urinary excretion of coproporphyrin I which are high during relapse, return to normal during remission(23). Changes in serum cholesterol and blood sugar has also been noted. The improvement induced in combined degeneration of the cord is believed to be due to vitamin B_{12}'s neurotrophic action and possibly its role in protein (21) and lipoid(24) metabolism causing reversal of early lesions in the nervous system. There is presumably a stage when the lesions are reversible when myelin is degenerating but axis cylinders are intact. Improvement is also due to greater use of unaffected pathways following arrest of lesions in the nervous system. In the peripheral nerves even destruction of axis cylinders is no bar to recovery, for new axons may grow down, or regeneration may occur.

SPECIFIC TESTS FOR PERNICIOUS ANEMIA WITHOUT TYPICAL ANEMIA: In searching for tests that would specifically prove the diagnosis of pernicious anemia in the absence of anemia, I have come to the conclusion that one must be aware of three-groups of findings associated with this disorder.

To the first group belong "tissue intrinsic factor" which apparently is present in the same concentration in the tissues and blood of healthy subjects and of pernicious anemia patients regardless of the hematological status (15). Here may also be included the *agent responsible for the binding capacity of the serum for vitamin* B_{12}. The estimation of these two factors, therefore, appears to carry no diagnostic value at the present state of our knowledge.

To the second group belong findings which parallel the morphologic changes in the peripheral blood and bone marrow; i.e. during hematologic relapse the concentration of *total and free vitamin* B_{12} *in the serum* is abnormal while during hematologic remission it is normal. Thus nothing will be gained diagnostically by estimating, with the Euglena gracilis var. bacillaris microbiological assay method, the concentration of total and free vitamin B_{12} in the serum of a pernicious anemia patient presenting no anemia. To this group also belong findings of *vitamin* B_{12} *concentration in the tissues* as well as *in the liver*. These findings, like the blood picture, will appear to be within normal limits in spite of the fact that the nerve lesions are progressing due to activity of the disease process. Findings of gastroscopic examination can also be included in this second group; for it is only during hematologic relapse that the gastrocopic picture is specific for this malady but not invariably during hematologic remission. Following liver(25) or vitamin B_{12} therapy, in some patients no improvement of the gross atrophic changes of the stomach mucosa is noted, in others it is partially improved, while in many patients the gastroscopic appearance of the mucosa is restored entirely to normal. The fact that in the latter cases there is apparently no definite proof of histological restitution — as pointed out by A. J. Cox who felt that the changes occasionally observed by the gastroscopist after treatment may be due only to the growth of replacement epithelium, or by Schindler who attributed the return of the normal gastroscopic picture, including the disap-

pearance of the visible blood vessels after B_{12} or liver extract to either a regeneration of the chief cells or to the formation of some unknown opaque nonspecific tissue element—still does not make one eliminate this diagnostic procedure from the second group in view of the gastroscopic findings.

The third group includes findings which are permanently and unconditionally elicited in pernicious anemia patients. Those findings are refractory to any known drug, and a state of maintained hematologic remission does not alter their abnormal condition as it does to the findings of the second group. To this group belong the findings of *histamine-resistant achlorhydria* and of marked *deficiency of "gastric intrinsic factor."* The first procedure is very popular and easily carried out. The estimation of the "gastric intrinsic factor" however, has met with great difficulties.

Castle's(10) classic bioassay consisting of feeding extrinsic factor, in form of beef muscle, mixed with gastric juice of an undiagnosed patient to a known pernicious anemia patient in hematologic relapse, and according to the response of the known pernicious anemia patient's hematologic system to judge about the status of the "gastric intrinsic factor" in the undiagnosed patient, is a very complicated and impractical procedure. This method has been known for 25 years, and needless to say it has rarely been put into practice.

Lately, a new approach has been developed *which indirectly measures* the presence or absence of sufficient gastrointestinal intrinsic factor. Radioactive cobalt incorporated in vitamin B_{12} when given orally to the pernicious anemia patient can be recovered in large part in the feces. Heinle and his associates(28) were the first to administer vitamin B_{12} labeled with radioactive cobalt 60 orally, to normal subjects and to patients with pernicious anemia. The amount of radioactivity which appeared in the feces was greater in patients with pernicious anemia than in normal subjects, but could be decreased by administration of a source of intrinsic factor in conjunction with the labeled vitamin. Halsted et ac. (29) measured fecal excretion of cobalt 60 – labeled vitamin B_{12} with a scintillation counter. They found that eleven normal persons excreted on the average 33 per cent of 0.5 microgram of orally administered radioactive vitamin B_{12}, while seven patients with pernicious anemia excreted, on the average 93 per cent in 10 tests, but when a source of intrinsic factor was administered with the test done in 4 patients, fecal excretion was decreased to an average of 38 per cent. More interesting was the finding that 11 patients who had had total gastrectomy, none of whom had macrocytic anemia, excreted on the average 87 per cent in 16 tests. When a source of intrinsic factor was administered with the test dose, an average of only 20 per cent was excreted in 14 tests.

At this point it should be mentioned that Rubin and Massey(30) noted a typical appearance of the exfoliated cellular material from the stomach of patients with pernicious anemia persisting despite prolonged therapy. However, this approach is still in the investigative stage. Also in the investigative stage is the interesting observation of Olmstead and Hirschboeck(31) who found low urorennin levels in the urine of 50 pernicious anemia patients even in hematologic remission. They assumed that the low excretion of uronennin resulted from the low rennin and pepsin activity of the gastric secretions in pernicious anemia. However, this finding is not specific for pernicious anemia but rather a manifestation of atrophy of the gastric mucosa which also occurs without pernicious anemia.

It appears that radioactive cobalt incorporated in vitamin B_{12} would be the most useful and most practical adjunct in the diagnosis of pernicious anemia without anemia, as it measures, indirectly, the gastrointestinal intrinsic factor deficiency. Its importance will still be greater when it will be applied to every case of histamine refractory achlorhydria, even prior to the onset of clinical manifestations of neuropathy; and when signs of deficiency of gastric intrinsic factor would be elicited, therapy could be undertaken prophylactically. A day will come when its use will be widespread, but until then it still remains that *in the absence of anemia, the clinical findings of subacute sclerosis in association with histamine-resistant achlorhydria plus response to therapy* are the only practical means of establishing a diagnosis of pernicious anemia.

CASE REPORTS: Two cases that presented subacute combined sclerosis with histamine-resistant achlorhydria without typical erythropoiesis of pernicious anemia are reported here:

CASE 1. M. M., 38 years old, white female, visited me in my office for the first time in November, 1951, complaining of "a back condition."

She stated that 6 years previously, after the confinement with her second child, she lifted the older child and the next day had a severe low back pain. Since then she had worn a brace on and off when she felt that her back became tired. On several occasions she went to a physician for general checkups, including blood counts, but except for the sprained back nothing of significance was found. She was advised to take vitamins, which she had been taking all the time.

About three months previously she felt extremely weak in her back and legs, and she had to drag her legs along when walking. During her office visit she complained of numbness, chiefly on the right side, but no radiating pain. She denied any gastric upsets, but stated that 2½ years previously her tongue had become swollen, and ever since then "the skin had come off the tongue," and she has had a soreness of her tongue.

The patient's family history was noncontributory. On examination she was a well developed and well nourished, right handed, young looking woman, wearing a body cast. Her general examination was negative. She appeared to have good color, but her tongue was definitely smooth and without any demonstrable papillae. Neurologically she showed possible slight general weakness of her musculature, especially in her lower extremity. No localized atrophies or muscular deficits were demonstrable. Reflex examination revealed upper abdominals present, lower abdominals absent; the knee jerks were exaggerated bilaterally; the ankle jerks showed an active response on the left side, a somewhat diminished ankle jerk on the right. Sensory examination to ordinary modalities revealed diminished pinprick perception in the distribution of the right external cutaneous nerve. Vibratory and position sense were definitely impaired, although not lost in the feet and toes. Plantar stimulation revealed poor planter flexion bilaterally though no true Babinski could be demonstrated. The soles of her feet were unusually sensitive.

Gastric analysis revealed histamine fast achlorhydria. Complete blood count was normal.

She was referred to a hematologist for bone barrow studies. However, after finding a normal peripheral blood, the hematologist felt there was no need to do a bone marrow smear. I subjected this patient to massive parental doses of B_{12} twice weekly; it took about 3-5 months until a noticeable symptomic improvement was noted. After six months there were definite signs of neurological improvement except for vibratory sense.

Patient continued on maintenance parenteral B_{12} up until 2 years ago, when she was referred to another internist (because I joined the V.A.) who discontinued those treatments for 3 months in order for her to undergo bone marrow studies by a hematologist. When the latter discovered a normal bone marrow smear, his dictum was that patient had no pernicous anemia and he strongly urged her to give up gunshot therapy. However, her condition deteriorated and she had to resort back to B_{12} injections, with good results. She has been on a maintenance regimen since.

CASE 2. W.D.P. This was a 71 year old white male who was admitted to the Veterans Administration Hospital, Whipple, Arizona, on July 13, 1953, complaining of numbness and tingling, mainly in the extremities, of about 3 years duration; and of walking difficulties of about 2 years duration. He stated that his legs felt weak and he sometimes could not control them. For fear that he might get off balance he walked no more than 1½ blocks. He also stated that he had had anemia for some years, the cause of which could not be determined, and that was also his main reason for seeking hospitalization.

Past history consisted of an appendectomy and herniorrhaphy many years previously. Since 1925 he had had bronchial asthma and bronchitis. He stated that in 1939 he had a peptic ulcer and recovered fully after one year's therapy with diet, amphojel and discontinuation of smoking. In 1947 he was treated at a hospital in Los Angeles, California, for unresolved pneumonia, at which time about 200 penicillin injections were administered.

He was hospitalized again in Los Angeles, in 1951 because of an acute abdominal episode which was thought to be a duodenal ulcer perforation, but which upon laparotomy showed a well healed ulcer in the anterior portion of the duodenum and moderate induration of the pancreas with small punctate nodules on the surface of the mesentery associated with a small amount of fat necrosis. A diagnosis of interstitial pancreatitis was made, and no additional procedures were performed.

His third hospitalization at the same hospital in Los Angeles in February, 1952, occurred at the suggestion of his private physician, because of "an anemia". The patient's anemia on the outside was never diagnosed by laboratory tests, but rather by history from his family physician. On admission his hemoglobin was 7 gms. and red cells 3.5 million, and hematocrit was 26. He was given blood transfusions and vitamin therapy and ferrous sulfate. Following the 15th day of hospitalization he showed uric acid of 10.6 mg.%; cephalin flocculation was 2 plus in 48 hours, thymol turbidity was 9.1 units, and bromsulfalein showed 2.4% dye at 45 minutes. Cholecystogram on admission showed a non-functioning gallbladder. Several weeks later his blood count and cholecystogram were found to be normal. He was discharged from there with diagnoses of: (1) Anemia, etiology unknown. (2) Hyperuricemia, etiology unknown. (3) Hepatic parenchymal disease. The additional diagnoses were: Small ventral hernia and benign prostatic hypertrophy.

Physical examination on admission to Veterans Administration Center, Whipple, revealed a well developed, well nourished white male of 71, who appeared in no acute distress. He appeared mentally clear and cooperative. Positive findings consisted of: Arcus senilis. Tongue was partially deprived of papillae; the anterior part was smooth and beefy red. There was tenderness in the region of the 6th and 10th ribs. Postoperative scar noted in the left chest region. Moist rales were elicited at both lower lung bases posteriorly. The right lung appeared to be more emphysematous than the left one. The point of maximum apical impulse was in the 5th interspace, one finger breadth beyond the midclavicular line. There was a soft apical systolic murmur, not transmitted to the axilla. P_2 was greater than A_2. Postoperative scars were noted over upper abdomen and bilateral inguinal regions. Pedal pulsations not elicited but extremities felt warm and oscillometric readings normal. The neurological examination revealed reflex changes. The knee jerks were slightly hyperactive. The ankle jerks were absent. Babinski and Oppenheim were positive. There was moderate ataxia, demonstrated on finger to nose and heel to knee tests. A moderately coarse tremor of the extended hands was noticed. The vibratory sense was moderately diminished over the lower extremities. The sensory changes were mainly subjective, consisting of dysesthesias and paresthesias involving not only the four extremities but to a lesser degree the entire body.

LABORATORY: July 13, 1953. Red blood count 3,200,000. Hemoglobin 8.9 grams. Hematocrit 32 mm. White blood count 10,500; Neutrophils 68, lymphocytes 28, monocytes 4, band cells 2. Platelets 275,000. Bone marrow aspiration showed 1 myeloblast; 30 myelocytes neutrophils; 1 myelocyte-eosinophil; 17 meta-myelocytes-neutrophils; 19 P.M.N. neutrophils; 2 lymphocytes; 1 monocyte; 3 megaloblasts; 26 normoblasts. M-E ratio 71-29. Cells counted 200. Sedimentation rate 22mm. Blood urea 17 mg.%. Glucose fasting 97 mg.%. Serum bilirubin 0.53 mg.%. Serum uric acid 4.8 mg.%. Urinalysis Color, yellow; reaction acid; specific gravity 1.014; albumin and sugar negative; bile negative 3-5 hyaline casts; rare white blood cell. Urine urobilinogen negative. Serology negative. Cephalin flocculation 3 plus in 24 hours and 4 plus in 48 hours. Bromsulfalein 8% retained in 45 minutes. ree hydrochloric acid absent with histamine. Stools negative for ova, parasites; trace of fat; no meat fibres. Bleeding time 1 minute. Coagulation time 5 minutes. Coombs test negative. Red cell fragility test normal. Blood culture showed no growth in 16 days. eRticulocyte counts during August ranging between 3.4 to 4.7 per cent, and on September 24, 1953, 6.7 per cent. Complete blood count on October 21, 1953, revealed 5,370,000 red blood cells. Hemoglobin 14.9 grams. Hematocrit 53 mm. Cephalin flocculation was 4 plus in 48 hours. Alaline phosphatase 10 units. Thymol turbidity less than 1 unit and bromsulfalein 5% retained in 45 minutes. Total protein 6.8; albumin 3.7; globulin 3.1. Electrocardiogram was within normal limits. X-ray of chest revealed that the left 6th rib was partially resected and there was considerable fibrosis in the left mid lung field. The apex of the left lung was emphysematous. The heart was shifted to the left and there were pleural pericardial adhesions along the left heart border. There was compensatory emphysema of the right lung. There appeared to be an old healed fracture of the left 10th rib. The heart size was within normal limits. Barium enema yielded no abnormal findings. GI series showed clover leaf deformity of the duodenal bulb and diverticulum of the second portion of the duodenum. Contrast material concentrated well in the gallbladder, which showed no evi-

dence of stones and appeared to empty satisfactorily. A mared sclerosis of the abdominal aorta was noted. Examination of the esophagus showed no evidence of varices. X-ray of the spine showed hypertrophic osteoarthropathy of the cervical and dorsal spine.

COURSE IN THE HOSPITAL: Even though the patient revealed no typical P.A. bone marrow; no marked megaloblastosis or giant metamyelocytes; and no characteristic changes of pernicious anemia in the peripheral blood picture, he was treated as a pernicious anemia case with B_{12} and liver injections and the results had been good. In December, 1953, patient was found to walk much better, had much less ataxia, and showed increase in general strngth and vigor. His dysesthesias and paresthesias improved greatly although reflex changes persisted. One month later patient revealed excellent coordination and stated that he felt much stronger in his legs and was able to walk for many blocks. Numbness and tingling had gone entirely. When he was sent on a therapeutic trial visit for a longer period of time and was off parenteral and only on oral medication of B_{12} plus intrinsic factor his neurological condition definitely deteriorated while the hematological remission persisted.

COMMENT: The first patient had taken vitamin preparations containing B_{12} and folic acid for several yaers. She presented subacute combined sclerosis and histamine resistant achlorhydria and glossitis, and at no time anemia. Undoubtedly her back sprain "masked" to some degree the subacute combined sclerosis and the absence of anemia "preventd" the diagnosis of pernicious anemia.

The second patient had on several occasions been subjected to prolonged antibiotic therapy and had taken vitamin preparation on and off until one year prior to his last hospitalization. He showed anemia but no bone marrow characteristic for pernicious anemia. Since the attack of acute pancreatitis in 1951 he has at no time thereafter revealed symptoms or signs compatible with pancreatitis: serum amylase, urine diastase, glucose tolerance curve, stool for meat fibres and the clinical findings all disproved pancreatitis. The abnormal liver function tests pointed to hepatocellular damage, which is not uncommon in pernicious anemia. So did Schilling and Harris(32) report that the bromsulfalein retention exceeded 5% in 45 minutes, and cephalin flocculation was 3 plus in 48 hours in 3 of 20 patients with pernicious anemia. Alkaline phosphatase was elevated in only one patient; and total serum protein concentrations were lower than the accepted range in all of their 20 patints. So also did this patient reveal a nonfunctioning gallbladder concomitant with the anemia in 1952, which then reverted to normal following blood transfusions and hematological remission. This is in accord with the report of Lindquist(33) who found that oral cholecystography failed to outline the gallbladder in 16 of 33 patients with pernicious anemia in relapse; repetition of the test in 12 of those patients after hematological remission had been induced showed definite visualization of the gallbladder in eight. Because of liver damage, arteriosclerosis, chronic bronchitis and emphysema in this patient reticulocytosis following massive B_{12} and liver injections was markedly delayed. The most interesting feature about this patient is the history of a duodenal ulcer. It proves that one may develop pernicious anemia following a peptic ulcer if the latter heals and hyperchlorhydria is substituted by histamine refractory achlorhydria. Absolutely true appears to be the reverse, namely that one who has pernicious anemia can hardly ever get a peptic ulcer because the histamine resistant achlorhydria of pernicious anemia is unconditionally permanent, and there can be no active peptic ulcer with achlorhydria.

SUMMARY: The increase in incidence of primary pernicious anemia without anemia because of ingestion of folic acid, vitamin B_{12} plus intrinsic factor incorporated into vitamin preparations on the market, increased parenteral administration of vitamin B_{12} in a host of conditions, and possibly because of antibiotics is stressed.

Subacute combined sclerosis in association with histamine resistant achlorhydria is rarely encountered in any other condition but pernicious anemia, and the differential diagnosis can, almost always, be established on the basis of clinical evidence alone.

Physiological and other changes associated with pernicious anemia are reviewed. The *findings common in pernicious anemia are classified into three groups on the basis of their diagnostic value especially in a state of morphologically normal peripheral blood and bone marrow.* Histamine resistant achlorhydria and deficiency of gastric intrinsic factor because they are uncon-

ditionally permanent findings in pernicious anemia and not influenced by hematological, or any other, remission, when demonstrated, specifically confirm the diagnosis.

The importance of radioactive cobalt labeled vitamin B_{12} as a diagnostic aid and its utilization as a preventative tool for pernicious anemia neuropathy in the future is suggested.

Two cases of pernicious enamia without typical erythropoiesis are reported. The delay of proper diagnosis and treatment in the face of absence of characteristic hematological findings while serious neurological lesions were progressing, is described. The second case, in addition is more intriguing as he proves that pernicious anemia may follow a peptic ulcer when the latter turns inactive and histamine-fast achlorhydria substitutes for hyperchlorhydria.

ADDENDUM: Since preparation of this paper one more case of postero-lateral sclerosis and histamine-refractory achlorhydria with a normal hematological picture was treated at the Medical Services of V.A.H. Whipple, with parenteral B_{12} with mared clinical improvement.

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THE GONORRHEA PROBLEM IN ARIZONA

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OCCASIONALLY a communicable disease which seems to be under control, or nearing control, flares up and we suddenly find we have a problem on our hands. This is true of gonorrhea. For the past several years the gonorrhea attack rates, as reflected by cases reported to the State Department of Health, have remained more or less steady. If a trend is to be noticed over the years, it would be slightly downward.

Suddenly, last July the gonorrhea cases reported began to increase. This, in itself, was not alarming as very often there is a sudden increase in cases of venereal disease reported, usually followed by a period of decreased activity. However, in this instance, the expected decrease has not materialized. Instead of decreasing, the reported cases of gonorrhea have continued to increase. This increase is shown by comparing the total number of cases reported in 1954 with those reported in 1953; there was an increase in 1954 of 50%.

This increase was not limited to any one area, but rather there was an obvious increase throughout the state. The trend established in 1954 has continued. During the first six months of 1955 a total of 490 more cases of gonorrhea has been reported than during the same period last year; an increase of 54 percent.

What is the meaning of this increase? Does it actually mean that there is a serious epidemic of gonorrhea, or can it be explained in some other way? First of all, could this increase be due to the more exact reporting of cases of gonorrhea by the physician rather than an actual increase in the number of cases detected? This might be true if only the private physician were involved, but the number of cases reported by local health units, hospitals and other institutions also increased. It is true that a number of physicians reported cases of gonorrhea for the first time in 1954. However, physicians who regularly report, generally reported more cases in 1954 than during 1953. Then too, no concentrated drive to get physicians to report cases of venereal disease had been carried out during the past year.

Second: Arizona is a growing state. Could this upward spiral of cases reported be merely the result of an increased population? The population of the state increased between 5 and 10 percent during 1954, while the gonorrhea cases reported increased 50%. This population increase hardly seems to be the answer.

Third: During the political campaign last summer and fall, Arizona's unfavorable health picture with its high venereal disease rates was explained to some extent in the campaign. Has this publicity made the population and the physician more venereal disease conscious, which has resulted in more people seeking treatment and being treated for gonorrhea? This could be true, although this same situation was publicized in other political campaigns without any increase in venereal disease being reported to the State Department of Health. If this supposition were correct, it would have resulted in an increase in the cases of syphilis reported too. However, this is not the case. The number of syphilis cases reported have actually gone down, not up, during this period.

The fourth supposition would be that there has actually been an increase in promiscuity which would naturally result in an increase in venereal diseases. There is a great deal of evidence to support this theory. First, one would not expect much change in the sex habits of those in the older age groups. They should be well established by the time the individual reaches 20 to 25 years of age. Therefore, any general increase in promiscuity should be detected first in the younger ages.

A comparison of the age distribution of gonorrhea cases reported during 1953 and 1954 shows that while there has been an increase in the cases reported for practically every age, the increase in the younger ages is more marked. In 1953 the median age of cases of gonorrhea reported was 22 years. In 1954 this age was 20 years. This is all the more remarkable when one considers that there are many physicians who are reluctant to report venereal disease among teen-agers.

A second indication that more teen-agers are contracting gonorrhea is that during the past six months several physicians have called the state and local health units because they have become

alarmed about this situation. This concern is something that had rarely happened before.

A fifth supposition that might account for our present situation is that we have not been able to reduce the female reservoir of gonorrhea patients, or prevent it from increasing. The technique used to reduce this reservoir or keep it in check is the interviewing of male cases of gonorrhea for their sexual contacts and then the tracing of these contacts and bringing them to diagnosis. It might be that we are failing to get the cases which are doing the damage, in spite of the fact that we have had more and better contact investigation the past year than in previous years.

Whatever the reason, the fact is we seem to have a sustained epidemic of gonorrhea. What can be done about it? There are several things that the private physician may do to help:

1. Interview all male cases of gonorrhea for their sexual contacts.
   (a) If the patient is married, do not treat the husband unless sure that the wife is treated at approximately the same time.
   (b) Report all contacts solicited to your local health unit if in Pima, Pinal or Maricopa Counties, or to the State Health Department if not in the above mentioned counties. These contacts will be traced and brought to diagnosis and treatment if it is necessary.
2. If the physician does not have time to interview the patient and the patient lives in Pima, Pinal or Maricopa County, a call to the local health unit will bring a trained investigator to do the job.
3. Treat all female contacts of known male cases whether or not the gonococcus can be demonstrated by laboratory procedures. It is sometimes very difficult to demonstrate gonorrheal infection in old chronic cervicitis cases; yet they are extremely infectious. These cases need not be reported unless the physician has convinced himself either through clinical or laboratory procedures that the person has gonorrhea. Neither does this person need be known as a case of venereal disease. However, if they are treated a case of infectious gonorrhea may have been treated and the spread of infection to others prevented.
4. Report all cases of known gonorrhea to the State Health Department. This will give us an idea of the actual prevalence of the disease and point up areas that need special treatment.

Next, what does this epidemic of gonorrhea mean as far as other venereal diseases are concerned? Why have not the syphilis cases kept pace with the gonorrhea cases reported? If there is an increase in promiscuity, then there should be an increase in syphilis cases also. It is estimated that about five percent of those who contract gonorrhea contract syphilis at the same time. It appears that we are not even finding these cases. Are gonorrhea patients returning monthly for four months after treatment for a blood test and examination? Does the physician in treating his gonorrhea patient give him enough penicillin to cure him of any syphilis he may have acquired at the same time? Or does he give him enough to take care of the gonorrhea and only mask the early symptoms of syphilis, but not cure it? All these questions can only be answered by the physician himself. However, the fact remains that suspicion of syphilis should be considerably sharpened in the mind of any physician who is treating cases of gonorrhea.

There are still several foci of spyhilitic infection in the state which have not been wiped out. We are still receiving a considerable number of reports of infectious syphilis which indicates that there is enough syphilis in the state so a promiscous person need not hunt too long to find it.

Health authorities have the uneasy feeling that maybe many physicians in the state believe that the syphilis problem is solved and there is no further need to try and diagnose it. It is hoped that this is not true. We hope that the present downward trend in cases of syphilis reported is actually a true decline in the incidence of this disease and that it might soon cease to be a public health problem in Arizona.

PHOENIX Clinical CLUB

The Case History in this discussion is selected from the Case Records of the Massachusetts General Hospital, and reprinted from the New England Journal of Medicine. The discussant under Differential Diagnosis is a member of the staff of the Massachusetts General Hospital. The other discussants are members of the Phoenix Clinical Club.

CASE RECORDS OF THE MASSACHUSETTS GENERAL HOSPITAL NUMBER 37521

PRESENTATION OF CASE

A FIFTEEN-month-old girl was admitted to the hospital because of jaundice.

One month prior to admission while cutting the bicuspids the child developed bilateral swelling under the chin, low-grade fever and some reddening of the right eardrum. She was treated with ear drops and a sulfonamide compound for three days, with subsidence of the symptoms. At about this time she was observed to be scratching her skin a great deal but otherwise appeared well. No jaundice was noted Six days prior to admission a red blotchy rash developed over her neck and face. She was afebrile. At this time the mother noticed that the sclerae had a yellowish tint and that the abdomen was becoming larger. In the following two days there was a rapid increase in jaundice. The patient's appetite during this period seemed to improve, and she was quite active. Two days prior to admission she was seen by a physician and placed on skimmed milk. Thereafter the stools, which previously had been somewhat loose but of normal color, became hard, pale and infrequent. No stools were noted on the day before admission.

The child had had a normal birth and neonatal period, with a birth weight of 6 pounds and 3½ ounces. The pregnancy had been uncomplicated. She had been breast fed for two weeks and then switched to a whole-milk and Karo Syrup formula and at about one year chopped foods were added to the diet. She received oleum percomorphum, 10 drops, until the age of thirteen months. She was immunized against diphtheria, pertussis and typhoid between her sixth and eighth months but was not vaccinated. For one month following the loss of the cord stump there was recurrent discharge at the umbilical site, which healed after treatment by repeated cauterizations.

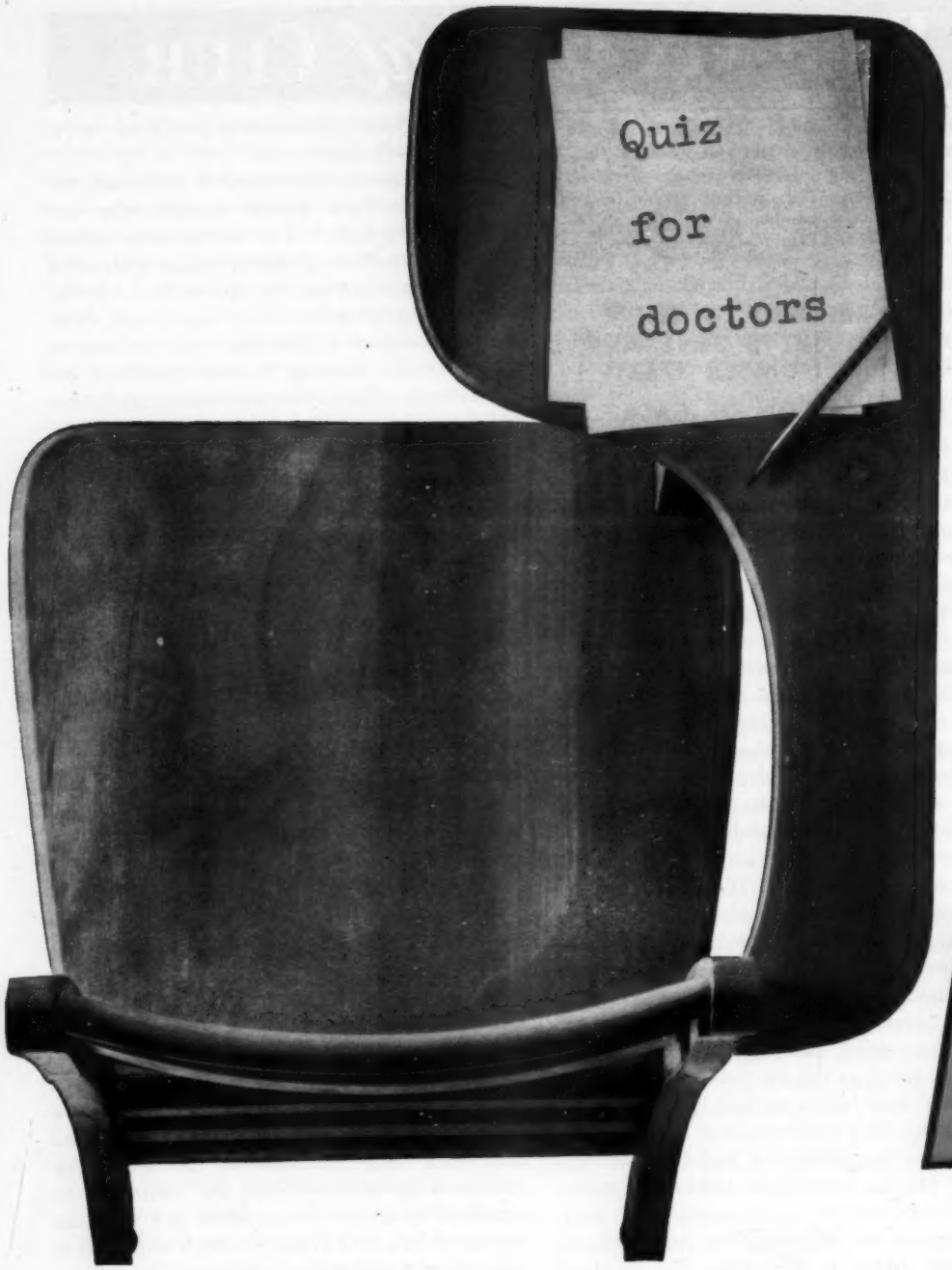
Physical examination revealed a 29 inch, 23-lb., well developed jaundiced child who was listless and irritable. The sclerae were yellow tinged. The skin was distinctly yellow with a few faint red macules at the upper chest. The pharynx was negative. The lungs were clear. The heart was of normal size with a Grade II apical systolic blowing murmur that was not transmitted. The abdomen was proturberant and a firm, smooth, nontender, 14x19 cm. mass was felt in the right upper quadrant that extended to the left of the midline. The mass extended into the right flank posteriorly and apparently occupied almost the entire right side of the abdomen. It was thought to be separate from the liver, which could be felt 2 finger breaths below the costal margin and had a sharp nontender edge. The spleen was not felt. No fluid was noted.

The temperature was 98.4°F., the pulse 116 and the respirations 48.

The urine gave a test ++ for albumin and a + test for bile; the sediment contained an occasional red cell and hyaline cast and 50 white cells per high-power field. Examination of the blood revealed a hemoglobin of 10.9 per cent lymphocytes and 5 per cent monocytes. On smear the red cells varied in size and there were many target cells and a rare stippled cetl. The prothrombin with 16 seconds, (normal 19.5 seconds).

X-ray films of the chest revealed clear lung fields with diaphragms elevated by a large intra-abdominal mass. Abdominal films showed a large soft-tissue mass present in the right side of the abdomen that had displaced the stomach and small and large intestime to the left. The mass was smoothly rounded and could not be separated from the liver shadow but could be separated from the kidneys, which appeared of normal size. Intravenous pyelogram showed the dye to be well concentrated, outlining nondilated urinary passages on both sides. There was a suggestion of a small amount of fluid in the peritoneal cavity.

The patient passed several clay-colored stools while in the hospital. Her condition remained


Quiz
for
doctors

(you probably know every answer!)

Q. Which is today's most widely prescribed broad-spectrum antibiotic?

A. ACHROMYCIN — it's first by many thousands of prescriptions.

Q. What are some of the advantages of ACHROMYCIN?

A. Wide spectrum of effectiveness.
Rapid diffusion and penetration.
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Q. Exactly how broad is the spectrum of ACHROMYCIN?

A. It has proved effective against a wide variety of infections, caused by Gram-positive and Gram-negative bacteria, rickettsia, and certain viruses and protozoa.

Q. In what way are ACHROMYCIN Capsules advantageous?

A. For rapid and complete absorption they are dry-filled, sealed capsules (a Lederle exclusive!) No oils, no paste...tamperproof.

Q. Who makes ACHROMYCIN?

A. It is produced — every gram — under rigid quality control in Lederle's own laboratories and is available only under the Lederle label.

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unchanged. She refused almost all food other than milk. On the third hospital day an operation was performed.

Dr. ROGER F. WHITE: This case presents so many conflicting and apparently contradictory aspects that I suspect the diagnosis must be a very simple one. Drs. Dysart and Randolph have covered the range of diagnostic possibilities very well; however, I will probably venture a third diagnostic possibility if I do not lose my courage as I work my way through this impromptu discussion.

The history of recurrent discharge at the umbilicus warrants consideration. Perhaps this was a manifestation of a patent urachus or a persistent vitelline duct. Such lesions, if they become cystic, rarely ever attain such a size as the mass reported in this case, and in any event would tend to be lower in the abdomen and more in the midline.

I do not believe that this mass represents a renal tumor. It is hard for me to conceive of a renal tumor of this size that would not have produced cachexia or manifested itself by an abnormal excretory urogram.

A neuroblastoma could present itself as the mass described in this case. It is just about the only tumor of childhood which could have attained such size in such a short period of time. However, if this were a neuroblastoma, I would again expect some cachexia, displacement of the kidney on the side affected, and some irregularity or nodularity of the mass.

It seems to me that the most salient feature in this case is the presence of jaundice and rather clear-cut clinical and laboratory evidence of obstructive jaundice. What sort of a lesion could produce both jaundice and an abdominal mass such as described? The mass is described as being firm and smooth, separate from the liver edge anteriorally but not in the lateral aspect. I suspect that the mass is due to a cystic dilation of the extrahepatic biliary duct system. The reason for the sudden appearance of this mass must be due to recent and acute obstruction. Whether this obstruction was due to torsion of a mass about the duct system or due to occlusion by a stone or a plug of inspissated bile is not at all clear. It would be very interesting to know whether this child had a chronic hemolytic anemia of the Cooley type; the report of many target cells in the peripheral smear cannot be ignored.

I have been asked to discuss this case extemporaneously, and I assume that such a request implies that I commit myself to a definitive diagnosis. I will submit that at surgery the operative findings revealed a cystic dilatation of the common bile duct or choledochus cyst.

DIFFERENTIAL DIAGNOSIS

Dr. CLAUDE E. WELCH: When I received this protocol several days ago I gave it to my twelve-year-old son to read over. I wondered if he could help me with the diagnosis inasmuch as he gets all his mdical information from the Reader's Digest. I asked, "What do you think it to be?" He said, "It is a cyst." I said, "What is in the right upper quadrant?" He replied, "The bile ducts." With that as a preliminary diagnosis I shall consider the possibility of a congenital cyst of the bile ducts, which is one of the commonest causes of these particular symptoms.

The history was not very characteristic or informative until just before the patient came into the hospital. The jaundice of six days' duration obviously had come on rather rapidly because nothing had been noted previously except a slight amount of scratching which suggested a low-grade jaundice. Although the presence of a previous acute infection and the sulfonamides are of interest, I do not think they are important to our discussion. Then the abdominal mass became apparent and there were clay-colored stools. The examination in the hospital gave objective evidence of jaundice and began to localize the disease. There was nothing remarkable in the past history. There was no suggestion of a chronic wasting disease. The child was well and her weight was approximately normal. She entered the hospital with a firm smooth non-tender mass in the right upper quadrant that extended across to the left side. The description of this mass is extremely important. I do not think I can expand on it as it obviously was described very carefully. There was no fever. The blood count was not too remarkable for a child of this age – that is, it certainly was not diagnostic. Except for the bile in the urine there was little evidence other than that the child was sick.

Dr. BENJAMIN CASTLEMAN: Here is a drawing of the mass made by the resident.

Dr. WELCH: In this drawing a large mass is shown extending down below the umbilicus, half-way over to the left hypochondrium and halfway down to the inguinal ligament and ap-

A Combined Neuro-Effector and Ganglion Inhibitor

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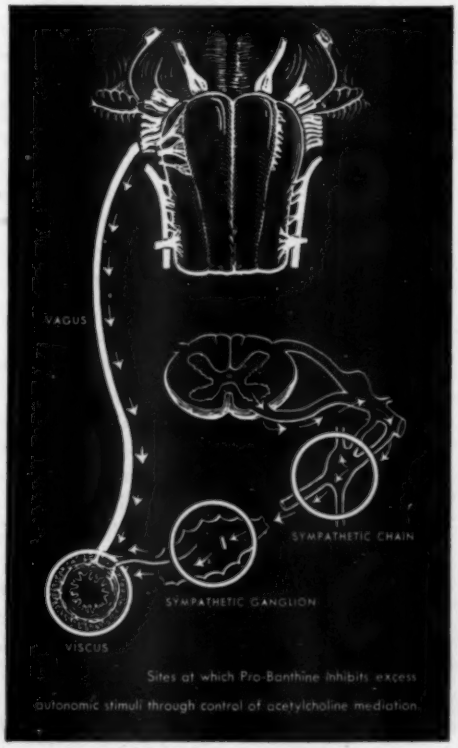

Sites at which Pro-Banthine inhibits excess autonomic stimuli through control of acetylcholine mediation.

Roback and Beal[2] found that Pro-Banthine orally was an "inhibitor of spontaneous and histamine-stimulated gastric secretion" which "resulted in marked and prolonged inhibition of the motility of the stomach, jejunum, and colon. . . ."

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In Roback and Beal's[2] series "Side effects were almost entirely absent in single doses of 30 or 40 mg. . . ."

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For the average patient one tablet of Pro-Banthine (15 mg.) with each meal and two tablets (30 mg.) at bedtime will be adequate. G. D. Searle & Co., Research in the Service of Medicine.

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1. Schwartz I. R.; Lehman, E.; Ostrove, R., and Seibel, J. M.: Gastroenterology *25*:416 (Nov.) 1953.
2. Roback, R. A., and Beal, J. M.: Gastroenterology *25*:24 (Sept.) 1953.

SEARLE

parently lying all anteriorly rather than posteriorly.

Being faced than with an abdominal mass in a child a litle over a year old, I have three major possibilities to consider. One is the presence of some maglinant tumor of which an embryoma of the kidney is the most common. There are other malignant tumors that I shall also have to consider. The second and third most common diagnoses are congenital cyst of the biliary tree and duplications of the intestinal tract – in this area particularly of the duodenum. With these three possibilities in mind, let us see the x-ray films.

Dr. JAMES J. McCORT: The palpable soft-tissue mass is seen filling almost the entire right abdomen and displacing the intestines to the left and downward. It is smooth in outline, lying somewhat anteriorly as described in the physical examination, and shows no calcification. Also it seems to be separate from the liver edge laterally, but anteriorly I cannot separate it from the shadow of the liver. A film of the abdomen was obtained in the lateral decubitus position to see if the mass would fall away from the edge of the liver and if there was any layering of fat within it. Neither of these was found.

On intravenous pyelogram the kidneys are normal in size, position and shape; they excreted the dye promptly and there was no obstruction to either urinary passage. The mass is entirely separate from the urinary track.

Dr. WELCH: There was no drink of barium I presume?

Dr. McCORT: NO.

Dr. WELCH: The x-ray findings are very helpful, particularly from the point of view of excluding a tumor of the renal tract. Let me consider first the possibility of some malignant tumor. Admittedly the differential diagnosis of a malignant tumor of children of this age is particularly difficult. If the patient had entered the hospital with the well marked cachexia that one might expect with a tumor of this huge size, then the diagnosis would be clear; but she was quite well developed and nourished, so I have no evidence from that point of view that she had a malignant tumor. Of malignant tumors that she may have had, a Wilms tumor is eliminated by the normal intravenous pyelogram. As I mentioned before, that is the commonest large malignant tumor in children. Years ago we had in this hospital a child of this age with a huge fibrosarcoma of the left lobe of the liver. As we know, other very bizzare tumors may appear in children of this age and may grow very rapidly. It is conceivable that this could have represented such a tumor, which was pressing up on the bile duct to produce the jaundice. This mass by x-ray examination seemed to be independent from the lobe of the liver and therefore a tumor originating in the liver can be ruled out. Another possibility that I ought to consider is a tumor arising from the sympathetic ganglia, such as a ganglioneuroma arising from the region of the right adrenal gland. Again there was no evidence in favor of that because there was no displacement of the kidney. I am not very well pleased with the diagnosis of malignant tumor.

I think it is more likely that I am dealing with a congenital abnormality. The most common congenital abnormalities that I have to consider are the congenital cysts of the bile duct and duplications of the duodenum. There is some evidence in favor of the cyst of the bile duct on several bases; one is the frequency of the two diseases. When Dr. Gross collected his first series of duplications of the duodenum, for example, he was able to find only one of those cases in the Children's Hospital records and was able to collect some 50 cases of congenital cysts of the bile ducts. Over ensuing years, congenital cysts of the bile duct are becoming considerably more common than duplication of the duodenum. The duodenum is one of the rarer parts of the intestinal tract to be duplicated. It is quite clear than any duplication would have had to be of the duodenum to explain the jaundice; that is the one tumor other than the cyst of the biliary tree that can produce jaundice.

Then I have some evidence from the symptomatology of the patient. When duplications of the duodenum occur (and cysts of the head of the pancreas and paraduodenal cysts all should be put in the same group). the patients usually have symptoms of duodenal obstruction. Jaundice is uncommon with the duplications. On the other hand, patients with cysts of the bile ducts have a triad of symptoms that consists of abdominal pain, jaundice and a mass. The pain was not well marked in this particular child, but it would be rather hard to elicit anyway. She was becoming irritable; that possibility represented a mild degree of pain. She certainly had a mass and she certainly had jaundice. Consequently from the point of view of symptomatol-

ogy this diagnosis would fit perfectly well.

I suppose she did have a cyst of the common duct; that of course could be manifested at her age. If these patients have a complete atresia of the bile ducts, the diagnosis is often made in the first two or three days of life. Dr. Nathan Talbot tells me this diagnosis is not always easy, since several days may elapse before jaundice is definite. If there is a simple cyst the diagnosis may be made at any time — even in adult life. It is of interest that one of the early patients on whom the diagnosis was made about twenty-five years ago is now in the hospital again. She has had repeated operations on the cyst of the common duct and now has a carcinoma of the head of the pancreas, perhaps indicating that there is more wrong congenitally than is suspected. Embryonic rests may be present as well as the cysts.

It is of interest to speculate on what influence the infection may have had on the onset of the symptoms. Many of these patients at operation or at autopsy are found to have patent ampullas of Vater. The infection may have been just enough to produce an obstruction at that site. Some of these patients have a condition that is probably analygous to an achalasia of the esophagus or colon in that everything appears to be perfectly normal except for the absence of nerve cells. At any rate, the etiology is far from clear. The treatment is more standardized. I believe that when the surgeon explored this girl he found a cyst of the bile duct that he anastomosed in some fashion to the intestinal tract.

CLINICAL DIAGNOSIS

Enterogenous cyst (duplication of duodenum)

DR. WELCH'S DIAGNOSIS

Congenital cyst of common bile duct.

ANTATOMICAL DIAGNOSIS

Congenital dilatation of common bile duca. (choledochal cyst).

Obstructive cirrhosis of the liver.

PATHOLOGICAL DISCUSSION

Dr. Castleman: I am sorry that Dr. Robert R. Linton, who operated on this child, cannot be here. His preoperative diagnosis was a duplication of the duodenum. At operation, he found a cyst of the common duct, as Dr. Welch predicted. This anomaly probably should not be called a choledochal cyst because it is really a part of the wall of the common bile duct that has become markedly distended apparently due to some congenital weakness in the wall. Gross and Ladd prefer to call it congenital dilatation of the common bile duct. The dilatation of the common duct contained over a liter of bile, which was removed through a window in the wall; bile was seen coming down from the liver through a very small lumen. The bile was drained through a T-tube and an anastomosis made with the jejunum. The liver at the time of operation was grossly cirrhotic and a biopsy showed moderate bile stasis and definite obstructive cirrhosis — fibrosis and bile-duct proliferation. The amount of bile-duct proliferation was much more than one would expect with the short period of jaundice that she apparently had.

The follow-up on this patient is interesting. She continued to be jaundiced for about two weeks; the jaundice then gradually decreased until about three and a half weeks after operation, when the stools became tan and the jaundice disappeared. She was sent home with the T-tube still in place, to be left there for about six weeks for adequate decompression. I understand from a surgical standpoint of view that in these cases no attempt should be made to remove the cyst.

Dr. Welch: They are impossible to remove and the patients have usually done very well following anastomosis.

Dr. Kranes: Where does the obstruction arise in these cases? In the site of entrance into the duodenum?

Dr. Castleman: Usually it is in the lower end just proximal to the ampulla; the weight of the dilated and full sac produces a kink in the common bile duct.

ADDENDUM

Dr. Robert R. Linton: I wish to compliment Dr. Welch on making the correct diagnosis. From the surgical point of view, the chief point of interest is the type of anastomosis that was utilized. In most cases in the literature a direct choledochoduodenostomy has been performed, whereas in this patient a choledochojejunostomy using the Roux-Y approach was performed. In the former, ascending biliary infection is frequently a serious complication. It was hoped with the type of anastomosis used in this case that it might be possible to prevent it. The drainage tube is still in place and it is our plan to have patient return in a month for a cholangiogram. Whether further surgery will be performed or not will depend a great deal on the findings at that time.

THE *President's* PAGE

THIS page will be devoted to excerpts from an Editorial of the Journal of the American Medical Association, published previously; however, because of its importance, it is felt that it contains some essential facts which may be read or re-read with some interest."

THE 1954 MEDICAL CARE DOLLAR

The long-term trends noted in the text — declines in the proportions of the total medical care expenditures for physicians' and dentists' services and rises in the proportions spent for hospital services and for all other medical care — continued in 1954. In 1954 physicians received 27.50 cents and the dentists, 9.20 cents from the medical care dollar; this is about 3/5ths of a cent less for physicians and 1/5th of a cent less for dentists than in (revised) 1953. (See table 3 which shows 28.15 cents and 9.43 cents, and chart IV in text.) The amount going for drugs and sundries also continued to decline; the 1954 portion of the medical care dollar going for this item was 15.40 cents, or 7/10th of a cent less than in 1953 (table 3 shows 16.15 cents). Three fourths of a cent more went for hospital services in 1954 than in 1953; the precise amount was 26.71 (table 3 shows 26.02) cents). Slightly greater was the increase in the amount going for all other medical care — 21.19 in 1954 as compared with 20.40 in 1953 (table 3 shows 20.06 cents). For the reader who would like to round these percentages and prepare his own "1954 Medical Care Dollar" similar to chart IV, the following distribution is suggested: Hospitals — 27c, Drugs — 15c, Other — 21c, Dentists — 9c, and Physicians — 28c.

A breakdown of the three items which comprise "all other medical care" expenditures, is illuminating. In 1954, the total dollars expended for "ophthalmic products and orthopedic appliances" actually decreased; consequently, the proportion of the medical care dollar decreased silghtly — by 2/5ths of a cent. The proportion spent for "other professional services" decreased only slightly. On the other hand, the amount spent for "medical care and hospitalization insurance" in 1954 incresed by $188 million over 1953. (This item is, essentially, premiums less claims with some allowance for reserves; it also includes unstated but comparatively small amounts paid to group health plans and as student fees for medical care.) This is 33% of the additional dollar amount spent for medical care and drugs in 1954. (The proportions of the 1952 and 1953 medical care dollar going for the insurance item were given in text table 3A as 7.89 cents and 8.87 cents, respectively; the revisions given here change these proportions to 8.06 and 9.06.) The 1954 portion of the medical care dollar spent for "medical care and hospitalization insurance" was 10.35 cents. This is more than a cent above (10.35 less 9.06) that expended on the same item in 1953; for the first time in medical economic history the amount spent by consumers for the health insurance overhead was a larger share of the medical care dollar than that received by dentists. When it is realized that these are the overhead costs of **having** voluntary health insurance, the reader may obtain some idea of how great the increase has been in insurance coverage for the consuming public.

In summary, there was a shift in the parts of the medical care dollar; about 1½ cents less went to physicians, dentists, and the suppliers of drugs and sundries, while 1½ cents more went to hospitals and to the administration of medical care and hospitalization insurance.

Harry E. Thompson, M.D.
President, Arizona Medical Association.

Editorial

ARIZONA MEDICINE
Journal of
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CONTRIBUTORS

The Editor sincerely solicits contributions of scientific articles for publication in ARIZONA MEDICINE. All such contributions are greatly appreciated. All will be given equal consideration.

Certain general rules must be followed, however, and the Editor therefore respectfully submits the following suggestions to authors and contributors:

1. Follow the general rules of good English, especially with regard to construction, diction, spelling, and punctuation.
2. Be guided by the general rules of medical writing as followed by the OURNAL OF THE AMERICAN MEDICAL ASSOCIATION. (See MEDICAL WRITING by Morris Fishbein).
3. Be brief, even while being thorough and complete. Avoid unnecessary words. Try to limit the article to 1500 words.
4. Read and re-read the manuscript several times to correct it, especially for spelling and punctuation.
5. Submit manuscript typewritten and double-spaced.
6. Articles for publication should have been read before a controversial body, e.g., a hospital staff meeting, or a county medical society meeting.

The Editor is always ready, willing, and happy to help in any way possible.

DON'T MISS THIS

PHOENIX and Arizona are in for a treat medically speaking, since they have the privilege of being the host for the 1955 annual meeting of the Southwestern Medical Association in cooperation with the second annual conference on recent advances in medicine of the Maricopa County Medical Society have arranged an outstanding program. Notes regarding this program will be found elsewhere in the Journal and every physician in the area has been mailed a copy of the preliminary program so that it would be pointless to repeat it here. We urge every physician in Arizona, many of whom already are members of the Southwestern Medical Association, to read the preliminary announcements of this program and we are sure that he will agree that it is outstanding and something which he will not want to miss. Make your plans now to attend.

There is another bonus attraction for every physician who is in the Phoenix area at that time. The Phoenix Executive's Club has engaged Dr. Ralph J. Gampbell to speak to them on Friday, November 18 on the subject of socialized medicine. The full announcement concerning this will be found elsewhere in the Journal but because his subject should be of considerable interest to the physicians attending the Southwestern Medical Association meeting, the Executive Club has decided to open this meeting to all of the doctors and their wives who do not have conflicting meetings for that evening and who may wish to attend. Dr. Campbell left England because he did not wish to continue practice under their system and has now had five years experience in practice in the United States and should have a message which will be interesting to us all.

Again we say DON'T MISS THIS!

AN INVITATION FROM THE PHOENIX EXECUTIVE'S CLUB

THE Phoenix Executive's Club has invited Dr. Ralph J. Gampbell to address them on the subject of socialized medicine Friday evening, November 18 at 7:00 P.M. This is a dinner meeting to be held in the Thunderbird Room of the Hotel Westward Ho. Because of the interest which Dr. Gampell's message will have for the medical profession, the Executive Club has opened this meeting to any physician and his wife and their friends who may wish to attend. Dr. Gampell has practiced medicine in Britain under the socialized system in effect there and has come to America to escape that system. He has now practiced for five years in the United States and is well qualified to contrast and compare the two systems of practice.

Those physicians and their wives and friends who wish to attend should send in their reservations immediately. Contact the secretary of the Executive's Club, Miss Jane Hudgins, 1305 E. McDowell Road, Phoenix, Ariz. The dinner is $5.00 per plate.

TOPICS OF *Current Medical* INTEREST

RX., DX., AND DRS.

By Guillermo Osler, M.D.

THE new SYNTHETIC STEROIDS continue to seem safer and more effective as more reports appear each week. The trade-names include 'Prednisone' (Pfizer), 'Meticorten' (Schering), et. al. . . . The advantages over cortisone and ACTH drugs are the scanty effect on the electrolytes, fluid balance, psyche, etc., and the more efficient action on certain types of arthritis, asthma, skin lesions, and various types of allergy. . . . The sed. rate more often drops to normal. The salt intake need not be limited. There is a lack of blood pressure effect. . . . The dose may be restricted to 50 mg. per day at the start; 35 mg. daily for a week; and an average maintenance of 15 mg. per day.

HAVE YOU BEEN CALLED A HERO lately? You may be one, tho possibly an unconscious-type hero. The J.A.M.A. has said that "It is a wise doctor who knows his own danger", and the routine medical hero is an accidental one who does work in which hazard is inherent. . . . The greater heroism is that of the M. D. who sees a danger and then exposes himself deliberately for the benefit of a patient, or a cause, or for science. The deliberate martyr role is one which never lacks applicants, tho quite often they don't survive the third act. . . . We aren't all like that, but it's pleasant to know that more than a few of our colleagues are.

A SENSE OF ADVENTURE is also refreshing to see. The most recent example is the shift from Texas to Ohio of a person who has sometimes been quoted in this column, Chauncey ('Sarge') Leake. . . . He is a fabulously versatile pharmacologist, physiologist, medical historian, dean of medicine, vice-president of the U. of Texas, author, voracious reader and abstracter, etc., etc. . . . He has now resigned, moved to Ohio State at Columbus, and is to work in basic sciences at the huge new Health Center there. . . . When an old firehorse hears the alarm, I guess he's got to go. . . . Dr. Leake starts off each monthly issue of his international one-sentence abstract with the phrase "Calling Attention To,—", which is what we are doing here.

One seldom sees an announcement from an M.D. which describes a pleasant change of location as perfectly as the one from Frank L. Meleney of New York. We have a special interest, since he has been a fine surgeon, terrific investigator of infections and chemotherapies, and he contributed to ARIZONA MEDICINE about five years ago. . . . The engraved notice gives his name, the initials of his four degrees, a comment that he is a founder of the American Board of Surgery, and his major teaching title, "Professor EMERITUS of Clinical Surgery, Columbia University." . . . Then, he announces the transfer of his practice to MIAMI, FLORIDA! . . . Peace. Peace. It's Wonderful!

Oldsters with BRONCHIECTASIS are never free of infection in their respiratory tract, says Pittman of Mass. Gen'l. Hospital. He advises protection from 'colds', which invariably cause bronchitis and patchy pneumonitis, by starting intramuscular injections of penicillin at the onset and daily doses for 5 to 10 days. . . . We'd like to see a comparison of this routine and one which uses oral Tetracyn or some such drug. The convenience and theoretical advantages would lead us to use the latter anyway.

One of the exhibits at the A.M.A. meeting in Atlantic City described a means for REDUCING TRANSFUSION REACTIONS. Stephen, Martin, and Bourgeois-Gavardin of Duke say that when Pyribenzamine solution is added to a unit of blood there is a significant reduction in the chance of allergic and pyrogenic reactions. . . . The incidence of allergic reactions in 1,500 patients given plain blood was 3.1 per cent, compared to 0.6 per cent in the 1,600 patients given blood containing the anti-histamine. The reduction in pyrogenic reactions was not as great but amounted to about 40 per cent. . . . Hemolysis was actually less in the AH series. . . . The dose was 25 mg. of Pyribenzamine in the first pint of blood, and none in later pints unless more than 12 units were used, or unless the interval between transfusions was more than 6 hours.

FECAL IMPACTION and severe constipation are tough problems which now have another method of therapy. Wilson and Dickinson of Ann Arbor have said that 'AEROSOL OT' can be used to make the scyballous material porous to water or mineral oil. . . . It can be given TID by mouth (2 cc of a 1% solution in milk or fruit juice) or by enema (5 cc of a 1% aerosol in 1 to 2 oz. of mineral oil). . . . There is no notable effect on fat or protein absorption; the aerosol is non-Toxic; and it can be used indefinitely without change in effect.

HBA FOR SECURITY

THE HOSPITAL BENEFIT Bulletin

Special Published Bi-monthly by Hospital Benefit Assurance, First Street at Willetta, Phoenix October, 1955

THE BOARD OF DIRECTORS

OF

HOSPITAL BENEFIT ASSURANCE

TAKE PLEASURE IN ANNOUNCING THE APPOINTMENT OF

DUKE R. GASKINS, M. D.

AS

MEDICAL DIRECTOR

WITH OFFICES AT THE COMPANY'S HOME OFFICE

FIRST STREET AT WILLETTA • PHOENIX, ARIZONA

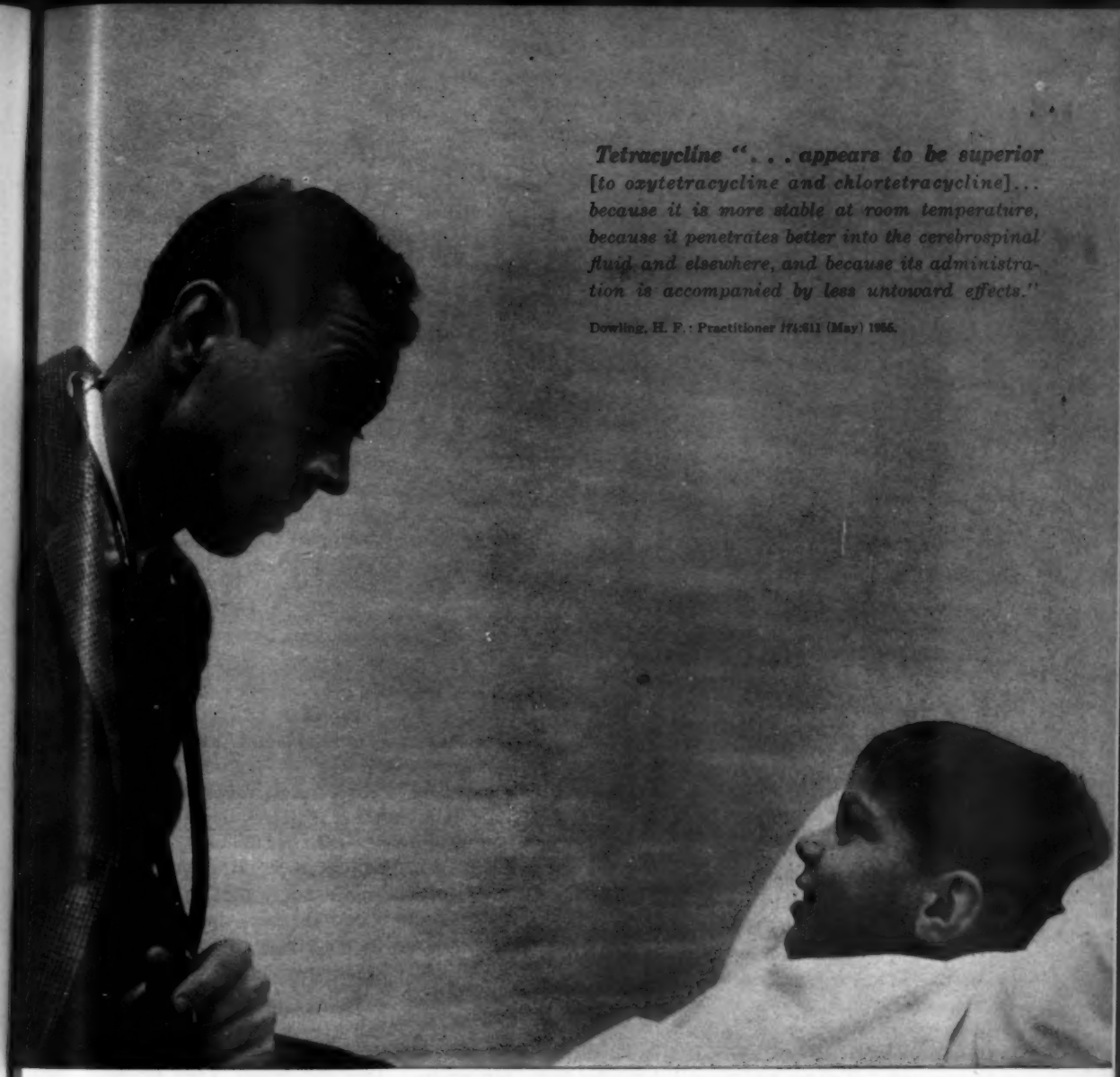
BRAVERY has an isomer which often occurs in patients. It is called COURAGE, and it may be of a positive, open sort (like when a patient faces pain or a gloomy diagnosis) or a quieter, hidden kind. . . . The QUIET courage is that which is required for awaiting the results of tests, or the courageous honesty which is needed to avoid cheating. . . . Why should a patient cheat, even in a negative way, when he is the one to suffer? Hard to say, unless you, yourself, have been presented with the chance. Most tests can't be faked or minimized (fortunately), but a sputum test for tubercle bacilli has less chance of being positive if one doesn't try very hard. Maybe it's brave to expectorate?

While mousing-around on the surface of patient and physician reactions it might be appropriate to mention a newly coined word,—'IATROERGIC'. . . . Most of us have barely become familiar with the similar term 'iatrogenic' (which means 'an effect or attitude which may be produced in the patient by the attitude of the physician'). . . . 'Iatroergic' is the effect on a physician of the pills he gives to the patient. It is a chief obstacle to honest research, even when controls are used, or placebos given in place of the drug being tested. The physician is so hopeful and convinced that he unconsciously influences the patient by the way he asks questions. . . . There can even by a 'negative iatroergy', due to the disenchantment of a physician with a mode of therapy.

The incidence of PSITTACOSIS is steadily increasing. Certainly the reportable incidence. There were 6 cases reported in the U.S. in 1941, 303 cases in 1954. . . . Fitz, Meiklejohn, and Baum of Denver describe 18 cases, conclude that ANY 'broad spectrum' antibiotic is good. . . . They advice I.V. therapy only for the sickest patients; use a priming dose of 2 grams; and a continued total of the same amount (in divided doses) per 24 hours. The patients usually are afebrile in 48 hours.

The ANABOLIC EFFECT of testosterone has been used to produce a weight gain in tuberculosis. It does not prevent healing, or surgical healing, and can be accompanied by anti-TB chemotherapy. . . . The dosage is 50 mg. three times a week, and the duration in the series reported by Griffith and Linn in the Amer. Review of TB was 6 weeks to 13 months.

DIAMOX isn't the answer to the problem of congestive heart failure, since it is somewhat toxic, but it is a good lead for the future. . . . It is a heterocyclic sulfonamide which induces acidosis by an alkaline urine. The action occurs by way of the inhibition of carbonic anhydrase in the renal tubules, followed by a lack of hydrogen ion from carbonic acid, an increased urinary excretion of sodium, a decreased ammonium excretion, an increased loss of potassium, and a decrease of edema due to diuresis. . . . A variable effect has been reported in cardiac decompensation, pulmonary emphysema, epilepsy, etc. Worth a greater trial, they say.



***Tetracycline** ". . . appears to be superior [to oxytetracycline and chlortetracycline]... because it is more stable at room temperature, because it penetrates better into the cerebrospinal fluid and elsewhere, and because its administration is accompanied by less untoward effects."*

Dowling, H. F.: Practitioner 174:611 (May) 1955.

excellent therapeutic response

with **Tetracyn®**

BRAND OF TETRACYCLINE

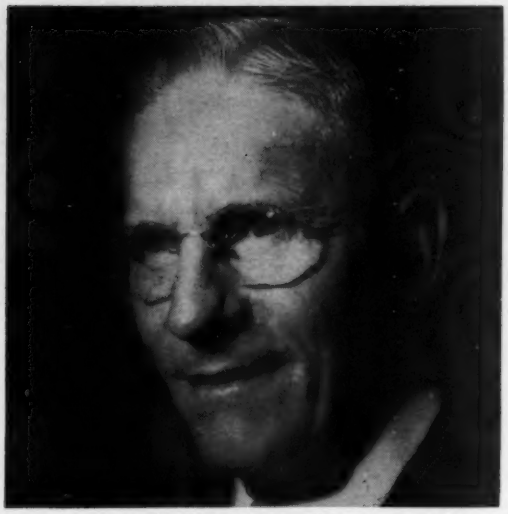
the original tetracycline

outstanding among modern broad-spectrum antibiotics

discovered and identified by **Pfizer**

Tablets and Capsules, 50, 100 and 250 mg., Oral Suspension (chocolate flavored), Pediatric Drops (banana flavored), Intravenous, and convenient ophthalmic and topical forms.

WHO CARES MOST



Fred G. Holmes, M.D.

FRED GOODING HOLMES, (1889-1955) departed this earth suddenly and tragically. Perhaps the manner in which he went makes more poignant the sense of evaluation of loss to those who knew him, and brings more sharply into focus the qualities from which we received our impressions of the man.

On Saturday, August 6, at five o'clock in the morning, he embarked with his son Fred W., and his two grandsons, Fred and Stephen, on the Klamath Bay, near the mouth of the Klamath River, in a sixteen foot boat. The boat was equipped with an outboard motor. It had life preservers for each, and salmon fishing equipment. Fred Sr. had been fishing the bay for some weeks prior to this fateful morning, and had been there a number of times in years past. They expected to return about 9 A.M. The fog rolled in before sun-up and nine o'clock passed with no sign of the party. Dense fog continued during the day. Search parties failed to locate any sign of the boat or the Holmes group. On the next day, the body of Fred Jr. was found washed up on the shore, about four miles north of the mouth of the Klamath River. Continued search up to the present writing, has failed to locate the rest of the members of the group. The boat was found awash near the body, and two life preservers were still attached. Whether outboard motor failure or confused directions as the result of the fog, swept them into the rough water of the river, where the tide meets the river flow, and capsized the boat, is speculation. The moments of fearful anguish can be imagined, facing certain destruction by the elements. To realize that communication was at end, when each had so much to say and so much service to render, must have been inexpressibly difficult.

Some of the facts of Fred G.'s life have particularly universal appeal. He not only worked his way through college, but at the same time helped support his mother and his brothers and sisters. Several times during our long association, Fred mentioned one of the things of which he was most proud, and that was that he had been number one Saturday Evening Post salesman, for the entire United States, during his years at Berkeley, University of California. One who has tried selling magazines knows that this accomplishment required a combination of tremendous energy, persistence in the direct approach, the ability to waste as little time as possible, to make moments count, to assume sincerity on the part of his casual contacts, and to demonstrate sincerity and honesty by his open simple appeal to prospects. The self-discipline and training thus acquired during his youth largely foretells the outstanding accomplishments of his mature years. He graduated from Harvard Medical School in time to serve in the First World War, following which he came to Phoenix, and started a general practice.

He called a consultation on a chest patient with one of the older doctors here, who demonstrated so much superior knowledge of chest diseases that the patient requested changing physicians. This challenged Fred G. to the point of sending him back to the Trudeau School of Tuberculosis, in 1920. From that time on his knowledge and skill in the treatment of chest diseases grew and his reputation extended. He was a great enthusiast for pneumothorax, which at that time was the most successful method of combating far-advanced tuberculosis. Several of the cases of bilateral pneumothorax, which he brought through extensive disease demonstrated for the first time how both lungs may be more than 75% collapsed, and still maintain satisfactory ventilation in the patient. On exhaling, under the fluoroscope, the lung shadows would entirely disappear behind the heart. Some of these early films were exhibited at the Century of Progress, Scientific Exhibit, in Chicago, in 1933.

In 1935, he called me into his office one day, and said, "Here is a film of a patient. What would you recommend?" Whereupon, I told him about six months to a year in bed. He said, "Well, that's my X-ray." During his bed rest, which turned out to be about nine months, he wrote a popular book for the patient on the subject of treatment for tuberculosis bringing it up to date on collapse therapy; he then published it at his own expense. While not a scientific treatise, this book was used quite widely, for a period of time in the sanitoriums around the country, as a means of assisting in the education of patients.

Voltaire wrote, "You seek for nothing but useful truths, and you say you have scarce found anything but useless errors." But Fred G. had a different philosophy and in that his simple faith gave him confidence in the rightness of his own conclusions. This facility may be said to be grounded in a deep informal religious faith.

At one time, he was all "steamed up" to run for State Senator, and thought he might have a good chance to be elected. However, he selected the U.S. Fair Employment Practices Act, which was being successfully discredited at the time, as his text for eight radio speeches, and soon after starting these talks, he found from the reaction of the electorate, that his chance of election was being dissipated. From that time on he continued making speeches with the idea that it was educational, in spite of the fact that he did not expect to be elected. He ran fifth in a field of six, but during the course of the campaign, his conviction that he was qualified and equipped to accomplish certain things in this life made him determined to quit the practice of medicine and devote his entire time to those ends.

One of these projects was the completion of the new and impressive Y.M.C.A. program for the City of Phoenix. This involved the collecting of a larger fund for charitable purposes than had ever been obtained in Phoenix before. Fred G. put his talents for salesmanship to work, helped to organize the campaign, and with tremendous energy assisted in most of the difficult tasks of persuading the various interests in the city to get together and really do a job in building this new Y.M.C.A. plant. Starting with the Board of Directors, he made it very clear that the only way these men could ask the community for money would be that they each and every one made sacrifices to give generously. He set the example and by persistence, the board followed him up to $60,000. Prospective givers were thoroughly "cased" and the key figures assisted him in going to see these givers in the most favorable circumstances. Along with a few kindred souls, they put over the first drive for funds and faced the disappointment of the shrinking dollar, in characteristic manner; repeating the drive and almost duplicating it. His persistent single purpose confidence was the ingredient, which added to that of several other individuals, made the community surprise itself.

A second theme, which occupied his enthusiastic attention, for a period, was brought about by his being invited to be an elder of the Presbyterian Church. While he had been a consistent valued worker in the church particularly in financial affairs, he entertained some doubts of signing the creed, without knowing fully what it contained. The fact that very little revision of the fundamental tenets of the church had taken place in 300 years challenged him to make an all out effort to have some of the basic parts of the creed revised. He wrote hundreds of letters and spent a considerable sum of his own money in trying to accomplish something nationally. He was elected to the fifteen man national board of the church with fourteen clergymen. He was not content to leave religious philosophy to the clergy. The question of the "damnation of unbaptized infants" was of real importance to him. But he was disappointed in the final outcome, as he was out-talked.

The final great sociological issue with which he greatly concerned himself was the question of discrimination against minority groups, within our social system. If one has any doubt about the value and importance of individual efforts or activities, the course Fred followed in regard to the minority groups should be a distinct and convincing lesson. Although the Supreme Court of the United States may not know it, Dr. Holmes' efforts in Arizona from 1948 on, may have profoundly affected the final decisions rendered in 1954, on the desegregation of schools.

Fred G. had placed himself in a position where he could not be greatly affected financially by adverse criticism. In this manner he had become a free agent to follow the dictates of his conscience without regard for the consequences

and his own economic welfare. Therefore, he was able to take the onus of some of the more difficult and disagreeable tasks in pointing out to the community the inconsistencies of discrimination. He accepted the brunt of the pro-discrimination attacks. His efforts materially stepped-up the time table for desegregation of the schools in the State of Arizona in 1953. This action by the state helped to create a "national trend", and could be interpreted as a swing of national opinion. It is generally accepted that the desegregation opinion of the U.S. Supreme Court was based as much on sociological data and public opinion as it was upon the law and precedent which indeed, was, to some extent, set aside. Fred G. so firmly believed that the very creation of an individual gave that individual the right to equality of opportunity that his mind worked incessantly to unravel the ramifications of the complex problem and simplify the steps which had to be taken, in order to make progress in the direction of accomplishment of that equality of opportunity. Chief Justice Warren is given responsibility for the Supreme Court action, but this rested squarely on what the country and the people were ready to accept. It is easier to deal with the abstract decision than to do some of the things Fred G. did. In these things he was a consistent, fearless, sincere, effective thinker – and doer.

Howell Randolph, M.D.

MEDICAL SUPPLY REPORT

OPERATION ALERT 1955 is over. It was assumed that atomic and hydrogen bombs had wrecked 58 cities throughout the nation. Approximately 8,200,000 people were killed, 6,500,000 were injured and vast areas were contaminated with radioactive fallout.

The nearest target city to the Phoenix area was Los Angeles, California, where three H-bombs hit the city on the morning of June 15th, left 797,000 dead, 637,000 seriously injured and demolished a third of the city's buildings.

Our supply problem was to assist the devastated area with the supplies available in this area, earmarked for civil defense use. Another and vital consideration was the care of thousands of evacuees pouring into Arizona, over two main highways, and arriving in a large mass at one time. This problem of supply made it essential that we make an immediate survey of drug resources and I believe that you will find the results of that survey interesting.

One shortage, of vitally important water purification tablets, was immediately obvious. Steps were taken at once to remedy this shortage and, as of this date, the necessary supply is on hand.

No attempt was made to ascertain the amount of narcotics available for such an emergency. I feel that some plan should be worked out with the Federal Government to insure a report on narcotics available for any disaster that might confront us in the future.

Another supply that is not considered at all in this report is that carried by retail pharmacies, which I believe are adequate.

Bob Shankland, pharmacist, has done a very fine job in regard to supplies at local pharmacies and should be highly commended for his untiring efforts.

We are very grateful for the complete cooperation of our drug jobbers, without whom this report to you would be impossible.

Our thanks to John Walker, Brunswig Drug Company, James Brandt, McKesson & Robbins Drug Company, and Emmett Davis, Phoenix Wholesale Drug Company.

MEDICAL SUPPLIES AVAILABLE FOR CIVIL DEFENSE

17.912	10cc vials	Penicillin G 300m units pr cc	
570	cc vials	Penicillin G 300m sgle. dose dispensable syringe	
440	5cc vials	Penicillin G 300m	
45	20cc vials	2 mil. u per cc Penicillin G Lilly	
25	20cc vials	5 mil. u per cc Penicillin G Lilly	

VASELINE in total lbs.

769 This item shows a great improvement over supply in 1954.

Burn Ointment, all types, in 1 lb. containers.

115 – plus 96 doz. Amertan & Unguentine in tubes.

Cotton – All sizes – Sterile	996 lbs.
Gauze bandage – all sizes	628 doz.
Gauze pads – all sizes	237 doz.
Adhesive tape – all sizes	61 doz.

Eye drops – all types 200 doz., plus 296 bots: Corton Acetate OPHT Sols.

Boric Acid Solution 4 oz. size, 125 doz.

Antibiotic Pediatric Drops – Sol. & Suspensions, also Syr. 8,937 bottles

Antiobiotic Caps & Tablets – for adult use, oral,

in bottles of 100's and includes Penicillin tabs, – 13,740, Terramycin, Aureomycin, Achromycin, Chloromycetin, Ilotycin & etc.

Sulfa drugs, all types in tablet form – in 100's – 7,611 bottles. This includes Bicillin, Pen Sulfa, etc.

Antiseptic's Tr Merthiolate	50 gals.
Diamond Antiseptic Tabs 7,000	
Listerine – all sizes	175 doz.
Zonite – all sizes	40 doz.
Bactine	30 gals.

Hypodermic Syringes – all sizes – 2½ gross.
Hypo needles – all sizes – 5 gross. This item is in short supply due to a strike at the Becton-Dickerson plant in New Jersey. This condition could be disasterous if an emergency should arise.

NOTE: This has now been corrected. First shipments from B.D. Co. came in this week.

This Report Dated – June 15th at 4 P.M. and submitted by:

Fred C. MacAlpine
Chief of Medical Supply
State of Arizona, Civil Defense

MEETING NOTICE

THE 1955 meetings of the WESTERN INSTITUTE ON EPILEPSY and the WESTERN SOCIETY OF ELECTRO-ENCEPHALOGRAPHY, cosponsored by the AMERICAN ACADEMY OF GENERAL PRACTICE (Ariz. Chapter) and the UNIVERSITY OF UTAH SCHOOL OF MEDICINE will be held in the Thunderbird Room of the Westward Ho Hotel in Phoenix on Thursday, Friday, and Saturday, November 10-12, 1955.

The program will include a seminar on the diagnosis and treatment of epilepsy and related convulsive disorders, clinical and research aspects of electro-encephalography, and presentations on the social, educational, and employment aspects of epilepsy.

Dr. Wilder Penfield

The guest of honor will be Dr. Wilder Penfield, Director of the Montreal Neurological Institute. Dr. Penfield's work in the field of epilepsy and cerebral localization is classical in the field of Medicine.

The committee, which is in charge of arrangements, includes: John R. Green, M.D., Phoenix, General Chairman, and President of both specialty groups; S. N. Berens, M.D., Seattle, Sec'y-Treas., Western EEG Society; Madison H. Thomas, M.D., Salt Lake City, Sec'y-Treas., Western Institute on Epilepsy; Matthew Cohen, M.D., and Robert A. Price, M.D., Phoenix, the Educational Committee of the American Academy of General Practice, Arizona Chapter.

All members of the Arizona Medical Association are cordially invited to register and attend the scientific and social sessions. Registra will begin at 3 P.M., November 9, 1955 and again at 8 A.M., November 10, 1955 in the Thunderbird Room of the Westward Ho Hotel, Phoenix. The first scientific session will begin at 9 A.M., November 10, 1955. The program has been approved by the Regional Commission on Education, American Academy of General Practice, for 6 hours of formal credit for members of the Academy.

SOUTHWESTERN MEDICAL ASSOCIATION

THE 1955 Annual Meeting of Southwestern Medical Association in association with The Second Annual Conference on Recent Advances in Medicine of the Maricopa County Medical Society, Phoenix, Arizona, November 16, 17 and 18, 1955, Hotel Westward Ho.

EMINENT GUEST SPEAKERS

H. L. BOCKUS, M.D.
Professor of Medicine, University of Pennsylvania Graduate School.

WILLIAM E. EHRICH, M.D.
Professor of Pathology, University of Pennsylvania Graduate School

L. KREER FERGUSON, M.D.
Professor of Surgery, University of Pennsylvania Graduate School

HANS. H. HECHT, M.D.
Associate Professor of Medicine, University of Utah

JOHN W. KIRKLIN, M.D.
Assistant Professor of Surgery, Mayo Foundation, University of Minnesota

I. V. PONSETI, M.D.
Associate Professor of Orthopedic Surgery, University of Iowa

KENNETH L. ROPER, M.D.
Associate Professor of Ophthalmology, Northwestern University

EDWARD H. RYNEARSON, M.D.
Chairman of Sections, Mayo Clinic

REGINALD H. SMART, M.D.
Clinical Professor of Medicine and Coordinator of Chest Diseases, University of Southern Cal.

JOHN WEBB, M.D.
Professor of Pharmacology, University of Southern California

J. WALTER WILSON, M.D.
Associate Professor of Dermatology, University of Southern California

PRELIMINARY PROGRAM

Symposium — Regional Enteritis and Enterocolitis — Doctors H. L. Bockus, L. Kreer Ferguson, and William E. Ehrich.

Symposium — Orientation of Massive Bleeding From the Upper Alimentary Tract — Doctors H. L. Bockus, L. Kreer Ferguson and William E. Ehrich.

Symposium — Experience with Subtotal Gastrectomy in the Management of Peptic Ulcer — Post-Gastrectomy Sequelae — Doctors H. L. Bockus, L. Kreer Ferguson and William E. Ehrich.

Prevention of Malignant Neoplasm of the Gastrointestinal Tract — Doctor H. L. Bockus.

Prophylaxis and Treatment of Complications Following Biliary Tract Surgery — Doctor L. Kreer Ferguson.

The Adrenals — Doctor William E. Ehrich.

Clinical Disturbances of the Pituitary Gland — Doctor Edward H. Rynearson.

Which Goiters Are Best Treated with Surgery and Which by Radioactive Iodine — Doctor Edward H. Rynearson.

Real Versus Supposed Disturbances of the Endocrine Glands — Doctor Edward H. Rynearson.

Symposium — Surgical Evaluation of Acquired Heart Disease — Doctors John W. Kirklin and Hans H. Hecht.

The Treatment of Atrial and Septal Defects — Doctor John W. Kirklin.

Surgery for Occlusion and Aneurysms of the Aorta — Doctor John W. Kirklin.

Essential Pulmonary Hypertension — Doctor Hans H. Hecht.

How to Evaluate Polycythemia — Doctor Hans H. Hecht.

Hip Disorders in Children — Doctor I. V. Ponseti.

Complications of Fracture Treatment — Doctor I. V. Ponseti.

Skeletal Lesions Produced by Aminonitriles — Doctor I. V. Ponseti.

Fungous Diseases — Doctor I. Walter Wilson

Delusion of Parasitosis — Doctor J. Walter Wilson.

The Effect of Drugs on Pulmonary Circulation and on the Control of Respiration — Doctor John Webb.

The Newer Drugs for Anesthesia — Doctor John Webb.

The Action of Drugs in Clinical Use for Heart Disease, Other Than Digitalis — Doctor John Webb.

Clinical Observations of the Value of Pulmonary Function Studies — Doctor Reginald H. Smart.

The Pneumoconioses and Pulmonary Function; Industrial Insurance Considerations — Doctor Reginald H. Smart.

Cataract Surgery — Doctor Kenneth L. Roper.

Ophthalmic Office Practice Procedure — Doctor Kenneth L. Roper.

Entertainment: Informal Party, Dinner Dance; special activities will be arranged for the ladies in attendance.

Hotel Reservations: Make hotel reservations at once through Chairman of Reservations, Maricopa County Medical Society, 2025 North Central Ave., Phoenix, Arizona.

We urge you to attend this outstanding meeting of the Southwestern Medical Association and the Annual Seminar of the Maricopa County Medical Society.

Joseph Bank, M.D., President, Southwestern Medical Association.

Carlos C. Craig, M.D., President, Maricopa County Medical Society.

Woman's AUXILIARY

SUMMARY OF AUXILIARY ACTIVITIES 1930 — 1955

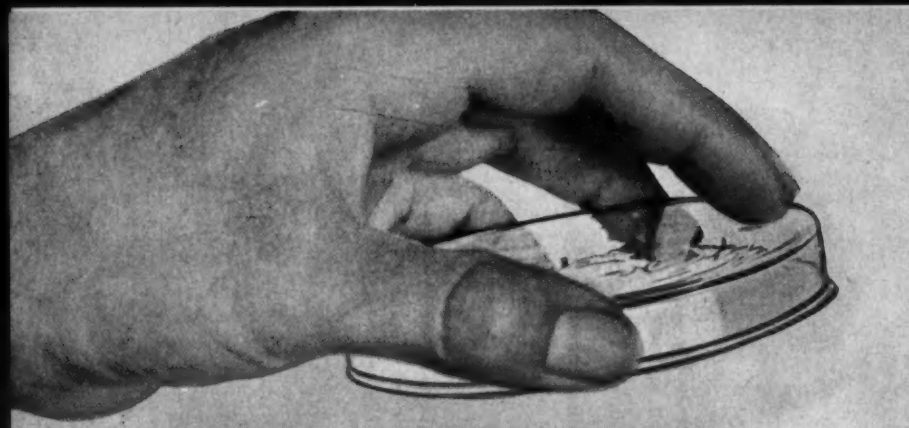
Mrs. Jesse D. Hamer
Phoenix, Arizona

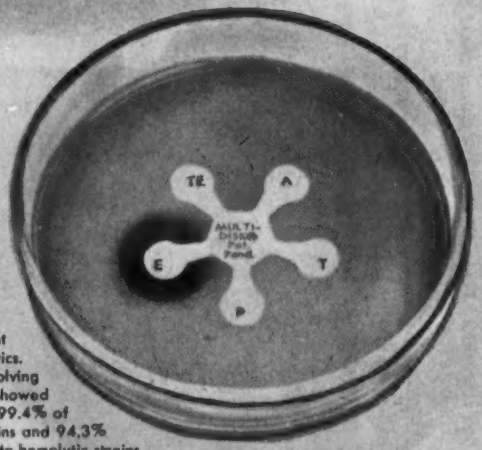
THE Woman's Auxiliary to the American Medical Association, during its 32nd annual meeting held at Atlantic City, New Jersey, June 6-10, 1955, conferred upon Mrs. Jesse D. Hamer of Phoenix, Arizona, Honorary Membership in that organization, which entitles her to all the rights and privileges of active membership.

Mrs. Hamer was president of the Woman's Auxiliary to the American Medical Association in 1946-1947; president-elect for two years, 1944-1946; and a director for four years, 1943-1944 and 1947-1950. She was chairman of the legislation committee, 1941-1942, and of the nominating committee, 1948-1949; western regional chairman of the war service committee, 1943-1944; a member of the reference committee for three years, 1948-1949 and 1952-1954, and of the finance committee, 1947-1950; and has been historian since 1947, a position she still holds.

In 1947, while president, Mrs. Hamer prepared a history of the first 25 years of the Woman's Auxiliary, which was published in the book called "A History of the American Medical Association", which is the official history of the American Medical Association and was compiled that year in connection with the organization's hundredth anniversary celebration. During the 1952-1953 auxiliary year, a 30 year history of the Woman's Auxiliary to the American Medical Association, prepared by Mrs. Hamer, was published in booklet form.

Mrs. Hamer is a charter member of the Woman's Auxiliary to the Arizona Medical Association, organized in 1930, and was its president in 1940-1941. She was president of the Woman's Auxiliary to the Maricopa County Medical Society in 1934-1935. For 25 years she has given continuous active service to the county and state auxiliaries.





DESTROYS ENTEROCOCCI

This blood agar plate shows a strain of *beta* hemolytic enterococcus. Note extreme sensitivity of this organism to ERYTHROCIN—yet it easily resists the other antibiotics. Additional data: A study[1] involving 202 enterococcic strains showed sensitivity to erythromycin in 99.4% of *alpha* hemolytic strains and 94.3% of *beta* hemolytic strains.

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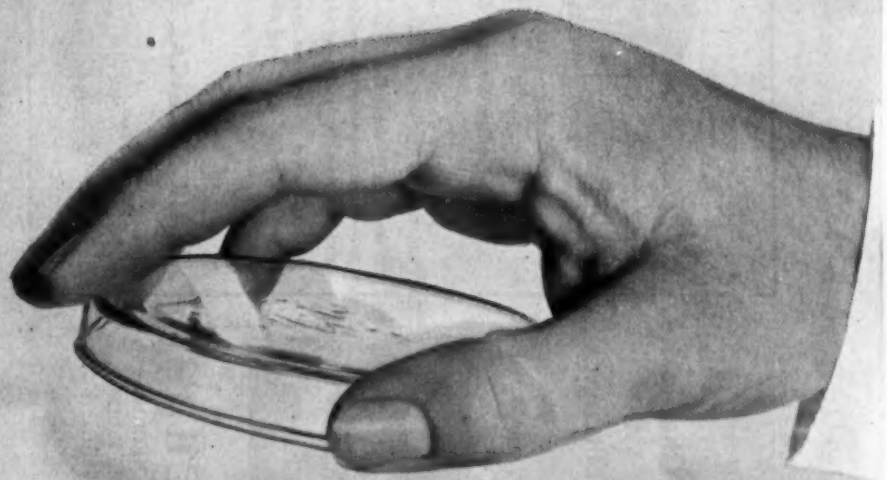

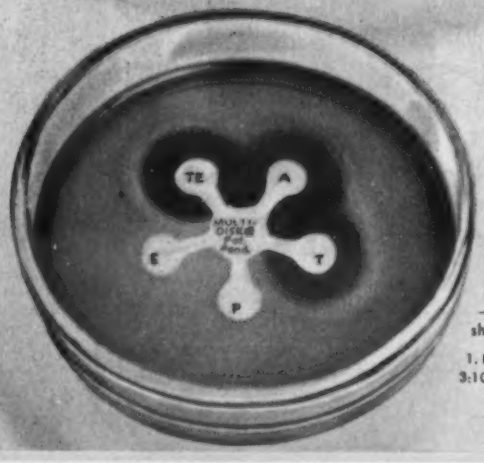

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This sensitivity test shows ERYTHROCIN and the same antibiotics against a typical intestinal strain of *E. coli*. Note that ERYTHROCIN and penicillin do not affect this gram-negative organism —although the other antibiotics show marked inhibitory action.

1. Eisenberg, et al., Antib. & Chemo., 3:1026-1028, Oct., 1953.

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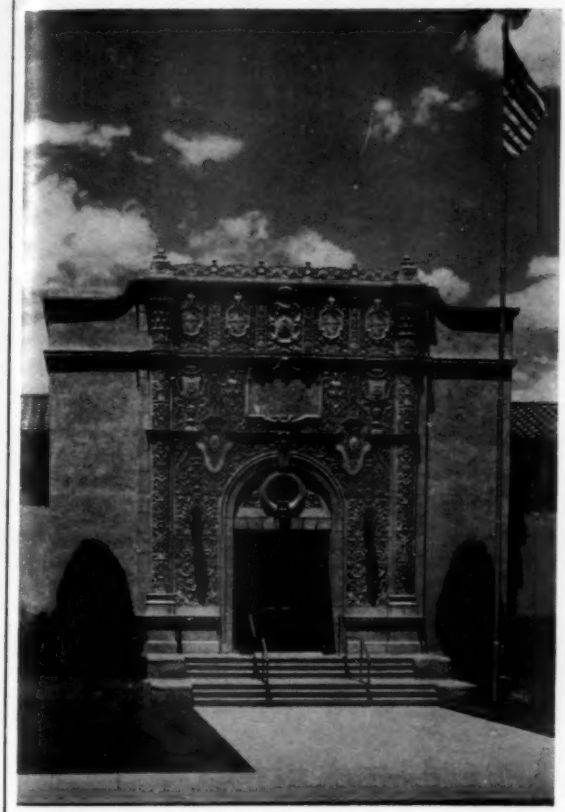

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